626

EXHIBIT

23a

Wind Turbine Syndrome

Nina Pierpont, MD, PhD

TWO

The Report, for clinicians

Abstract

This report documents a consistent and often debilitating complex of symptoms experienced by adults and children while living near large industrial wind turbines (1.5–3 MW). It examines patterns of individual susceptibility and proposes pathophysiologic mechanisms. Symptoms include sleep disturbance, headache, tinnitus, ear pressure, dizziness, vertigo, nausea, visual blurring, tachycardia, irritability, problems with concentration and memory, and panic episodes associated with sensations of internal pulsation or quivering that arise while awake or asleep.

The study is a case series of 10 affected families, with 38 members age <1 to 75, living 305 m to 1.5 km (1000 to 4900 ft) from wind turbines erected since 2004. All competent and available adults and older teens completed a detailed clinical interview about their own and their children's symptoms, sensations, and medical conditions a) before turbines were erected near their homes, b) while living near operating turbines, and c) after leaving their homes or spending a prolonged period away.

Statistically significant risk factors for symptoms during exposure include pre-existing migraine disorder, motion sensitivity, or inner-ear damage (pre-existing tinnitus, hearing loss, or industrial noise

628

exposure). Symptoms are not statistically associated with pre-existing anxiety or other mental health disorders. The symptom complex resembles syndromes caused by vestibular dysfunction. People without known risk factors are also affected.,

The proposed pathophysiology posits disturbance to balance and position sense when low frequency noise or vibration stimulates receptors for the balance system (vestibular, somatosensory, or visceral sensory, as well as visual stimulation from moving shadows) in a discordant fashion. Vestibular neural signals are known to affect a variety of brain areas and functions, including spatial awareness, spatial memory, spatial problem-solving, fear, anxiety, autonomic functions, and aversive learning, providing a robust neural framework for the symptom associations in Wind Turbine Syndrome. Further research is needed to prove causes and physiologic mechanisms, establish prevalence, and explore effects in special populations, including children. This and other studies suggest that safe setbacks will be at least 2 km (1.24 mi), and will be longer for larger turbines and in more varied topography.

Introduction and Background

Policy initiatives in the United States and abroad currently encourage the construction of extremely large wind-powered electric generation plants (wind turbines) in rural areas. In its current format, wind electric generation is a variably regulated, multi-billion-dollar-a-year industry. Wind turbines are now commonly placed close to homes. Usual setbacks in New York State, for example, are 305–457 m (1000–1500 ft) from houses.[1] Developer statements and preconstruction modeling lead

[1] Town of Ellenburg, NY, wind law: 1000 ft (305 m); Town of Clinton, NY, wind law: 1200 ft (366 m); Town of Martinsburg, NY, wind law: 1500 ft (457 m). For other examples in and outside NY State, see *Wind Energy Development: A Guide for Local Authorities in New York*, New York State Energy Research and Development Authority, October 2002, p. 27. http://text.nyserda.org/programs/pdfs/windguide.pdf.

communities to believe that disturbances from noise and vibration will be negligible or nonexistent.[2–4] Developers assure prospective communities that turbines are no louder than a refrigerator, a library reading room, or the rustling of tree leaves which, they say, easily obscures turbine noise.[5]

Despite these assurances, some people experience significant symptoms after wind turbines are placed in operation near their homes. The purpose of this study is to establish a case definition for the consistent, frequently debilitating, set of symptoms

---

[2] "The GE 1.5 MW wind turbine, which is in use in Fenner, New York, is generally no louder than 50 decibels (dBA) at a distance of 1,000 feet (the closest we would propose siting a turbine to a residence). Governmental and scientific agencies have described 50 dBA as being equivalent to a 'quiet room.' Please keep in mind that these turbines only turn when the wind blows, and the sound of the wind itself is often louder than 50 dBA. Our own experience, and that of many others who live near or have visited the Fenner windfarm, is that the turbines can only be heard when it is otherwise dead quiet, and even then it is very faint, especially at a distance." Letter from Noble Environmental Power, LLC, to residents of Churubusco (Town of Clinton), New York, 7/31/2005.

[3] "Virtually everything with moving parts will make some sound, and wind turbines are no exception. However, well-designed wind turbines are generally quiet in operation, and compared to the noise of road traffic, trains, aircraft, and construction activities, to name but a few, the noise from wind turbines is very low.... Today, an operating wind farm at a distance of 750 to 1,000 feet is no noisier than a kitchen refrigerator or a moderately quiet room." Facts about wind energy and noise. American Wind Energy Association, August 2008, p. 2. www.windturbinesyndrome.com/?p=698.

[4] "In general, wind plants are not noisy, and wind is a good neighbor. Complaints about noise from wind projects are rare, and can usually be satisfactorily resolved." Facts about wind energy and noise. American Wind Energy Association, August 2008, p. 4. www.windturbinesyndrome.com/?p=698.

[5] "Outside the nearest houses, which are at least 300 metres away, and more often further, the sound of a wind turbine generating electricity is likely to be about the same level as noise from a flowing stream about 50–100 metres away or the noise of leaves rustling in a gentle breeze. This is similar to the sound level inside a typical living room with a gas fire switched on, or the reading room of a library or in an unoccupied, quiet, air-conditioned office.... Even when the wind speed increases, it is difficult to detect any increase in turbine sound above the increase in normal background sound, such as the noise the wind itself makes and the rustling of trees." Noise from wind turbines: the facts. British Wind Energy Association, August 2008. www.windturbinesyndrome.com/?p=698.

630

experienced by people while living near wind turbine installations, and to place this symptom complex within the context of known pathophysiology. A case definition is needed to allow studies of causation, epidemiology, and outcomes to go forward, and to establish adequate community controls.

This set of symptoms stands out in the context of noise control practice. George Kamperman, P.E., INCE Bd. Cert., past member of the acoustics firm Bolt, Beranek and Newman (USA), wrote, "After the first day of digging into the wind turbine noise impact problems in different countries, it became clear that people living within about two miles from 'wind farms' all had similar complaints and health problems. I have never seen this type of phenomenon [in] over fifty plus years of consulting on industrial noise problems. The magnitude of the impact is far above anything I have seen before at such relatively low sound levels. I can see the devastating health impact from wind turbine noise but I can only comment on the physical noise exposure. From my viewpoint we desperately need noise exposure level criteria."[6]

I named this complex of symptoms "Wind Turbine Syndrome" in a preliminary fashion in testimony before the Energy Committee of the New York State Legislature on March 7, 2006. My observation that people can feel vibration or pulsations from wind turbines, and find it disturbing, was quoted in the brief section, "Impacts on Human Health and Well-Being" in the report *Environmental Impacts of Wind-Energy Projects* of the National Academy of Science, published in May 2007. No other medical information was cited in this report. The authors asked for more information to better understand these effects.[7]

[6] George Kamperman, personal communication, 2/21/2008. See www.kamperman.com/index.htm.

[7] National Research Council. 2007. *Environmental Impacts of Wind-Energy Projects*. The National Academies Press, Washington, DC. 185 pp, p. 109.

Debates about wind turbine–associated health problems have been dominated to date by noise control engineers, or acousticians, which is problematic in part because the acoustics field at present is dominated by the wind turbine industry,[8] and in part because acousticians are not trained in medicine. A typical approach to wind turbine disturbance complaints, world-wide, is *noise first, symptoms second*: if an acoustician can demonstrate with noise measurements that there is no noise considered significant in a setting, then the symptoms experienced by people in that setting can be, and frequently are, dismissed. This has been the experience of seven of the ten families in this study in the United States, Canada, Ireland, and Italy.[9] At least one developer has put forward the hypothesis that a negative attitude or worry towards turbines is what leads people to be disturbed by turbine noise.[10]

---

[8] George Kamperman, personal communication, 2/23/2008.

[9] A notable exception to this pattern is the physics research and modeling of GP van den Berg, who, as a graduate student and member of the Science Shop for Physics of the University of Groningen in the Netherlands, investigated noise complaints near a windplant and devised new models of atmospheric noise propagation to fit the phenomena he observed. References: 1) van den Berg, GP. 2004. Effects of the wind profile at night on wind turbine sound. J Sound Vib 277: 955–70; 2) van den Berg, GP. 2004. Do wind turbines produce significant low frequency sound levels? 11th International Meeting on Low Frequency Noise and Vibration and Its Control, Maastricht, Netherlands, August 30 to September 1, 8 pp.; 3) van den Berg, GP. 2005. The beat is getting stronger: the effect of atmospheric stability on low frequency modulated sound of wind turbines. J Low Freq Noise Vib Active Contr 24(1): 1–24; 4) van den Berg, GP. 2006. The sound of high winds: the effect of atmospheric stability on wind turbine sound and microphone noise. PhD dissertation, University of Groningen, Netherlands. 177 pp. http://irs.ub.rug.nl/ppn/294294104

[10] "We often use the word 'noise' to refer to 'any unwanted sound.' It's true that wind turbines make sounds ... but whether or not those sounds are 'noisy' has a lot to do with who's listening. It's also worth noting that studies have shown [no references provided in source document] that a person's attitude toward a sound—meaning whether it's a 'wanted' or 'unwanted' sound—depends a great deal on what they think and how they feel about the source of the sound. In other words, if someone has a negative attitude to wind turbines, or is worried about them, this will affect how they feel about the sound. However, if someone has a positive attitude toward wind energy, it's very unlikely that the sounds will bother them at all." Wind fact sheet #5: Are modern wind turbines noisy? p. 2. Noble Environmental Power, LLC. www.windturbinesyndrome.com/?p=698.

632

A reorientation is in order. If people are so disturbed by their headaches, tinnitus, sleeplessness, panic episodes, disrupted children, or memory deficits that they must move or abandon their homes to get away from wind turbine noise and vibration, then that noise and vibration is by definition significant, because the symptoms it causes are significant. The role of an ethical acoustician is to figure out what type and intensity of noise or vibration creates particular symptoms, and to propose effective control measures.

My study subjects make it clear that their problems are caused by noise and vibration. Some symptoms in some subjects are also triggered by moving blade shadows. However, I do not present or analyze noise data in this study, because noise is not my training. (Conversely, symptoms and disease are not the training of acousticians.) I focus on detailed symptomatic descriptions and statistical evaluation of medical susceptibility factors within the study group. Correlating the noise and vibration characteristics of the turbine-exposed homes with the symptoms of the people in the homes is an area ripe for collaboration between medical researchers and independent noise control engineers.

Other than articles on the Internet, there is currently no published research on wind turbine–associated symptoms. A UK physician, Dr. Amanda Harry, whose practice includes patients living near wind turbines, has published online the results of a checklist survey, documenting specific symptoms among 42 adults who identified themselves to her as having problems while living 300 m to 1.6 km (984 ft to 1 mi) from turbines.[11] She found a high prevalence of sleep disturbance, fatigue, headache, migraine, anxiety, depression, tinnitus, hearing loss, and palpitations. Respondents described a similar set of symptoms and many of the same experiences that

[11] Harry, Amanda. 2007. Wind turbines, noise, and health. 32 pp. www.windturbinenoisehealthhumanrights.com/wtnoise_health_2007_a_barry.pdf

I document in this report, including having to move out of their homes because of symptoms. Respondents were mostly older adults: 42% were age 60 or older, 40% age 45–60, 12% age 30–45, and 5% age 18–30. A biomedical librarian, Barbara Frey, working with this physician and others, has published online a compilation of other personal accounts of symptoms and sensations near wind turbines.[12] These also mirror what I document.

Robyn Phipps, PhD, a New Zealand scientist specializing in health in indoor environments, systematically surveyed residents up to 15 km (9.3 mi) from operating wind turbine installations, asking both positive and negative questions about visual, noise, and vibration experiences.[13] All respondents (614 or 56% of the 1100 households to whom surveys were mailed) lived at least 2 km (1.24 mi) from turbines, with 85% of respondents living 2–3.5 km (1.24–2.2 mi) from turbines and 15% farther away. Among other questions, the survey asked about unpleasant physical sensations from turbine noise, which were experienced by 2.1% of respondents, even at these distances. Forty-one respondents (6.7%) spontaneously telephoned Dr. Phipps to tell her more than was asked on the survey about their distress due to turbine noise and vibration, nearly all (39) with disturbed sleep.[14] Symptoms were not further differentiated in this study, but clearly may occur at distances even greater than 2 km (1.24 mi) from turbines.

Published survey studies have examined residents' reactions to wind turbines relative to modeled noise levels and visibility of

---

[12] Frey, Barbara J, and Hadden, Peter J. 2007. Noise radiation from wind turbines installed near homes: effects on health. 137 pp. www.windturbinenoisehealthhumanrights.com/wtnhhr_june2007.pdf.

[13] Phipps, Robyn. 2007. Evidence of Dr. Robyn Phipps, in the matter of Moturimu wind farm application heard before the Joint Commissioners, March 8–26. Palmerston North, New Zealand. 43 pp. www.wind-watch.org/documents/wp-content/uploads/phipps-moturimutestimony.pdf.

[14] Phipps 2007.

634

turbines in Sweden[15–17] and the Netherlands.[18–20] The study in the Netherlands included questions on health, though not of sufficient power to make any statements on health other than the correspondence between sleep disturbance and modeled noise (see below, Discussion). Both sets of studies, the Swedish and Dutch, have findings that could contribute to the rational setting of noise limits near wind turbines (see Discussion).

With regard to official opinion, the National Academy of Medicine in France recommended in 2005 that industrial wind turbines be sited at least 1.5 km (0.93 mi) from human habitation due to health effects of low frequency noise produced by the turbines.[21]

Current wind turbines have three airfoil-shaped rotor blades attached by a hub to gears and a generator, which are housed in a bus-sized box (nacelle) at the top of a nearly cylindrical, hollow

---

[15] Pedersen E, Persson Waye K. 2004. Perceptions and annoyance due to wind turbine noise: a dose-response relationship. *J Acoust Soc Am* 116(6): 3460–70.

[16] Pedersen E. 2007. Human response to wind turbine noise: perception, annoyance and moderating factors. PhD dissertation, Occupational and Environmental Medicine, Department of Public Health and Community Medicine, Göteborg University, Göteborg, Sweden. 86 pp.

[17] Pedersen E, Persson Waye K. 2007. Wind turbine noise, annoyance and self-reported health and wellbeing in different living environments. Occup Environ Med 64(7): 480–86.

[18] Pedersen E, Bouma J, Bakker R, van den Berg GP. 2008. Response to wind turbine noise in the Netherlands. J Acoust Soc Am 123(5): 3536 (abstract).

[19] van den Berg GP, Pedersen E, Bakker R, Bouma J. 2008. Wind farm aural and visual impact in the Netherlands. J Acoust Soc Am 123(5): 3682 (abstract).

[20] van den Berg GP, Pedersen E, Bouma J, Bakker R. 2008. Project WINDFARMperception: visual and acoustic impact of wind turbine farms on residents. Final report, June 3. 63 pp. Summary: http://umcg.wewi.eldoc.ub.rug.nl/FILES/root/Rapporten/2008/WINDFARMperception/WFp-final-summary.pdf. Entire report: https://dspace.hh.se/dspace/bitstream/2082/2176/1/WFp-final.pdf.

[21] Académie nationale de médecine de France. 2006. "Le retentissement du fonctionnement des éoliennes sur la santé de l'homme, le Rapport, ses Annexes et les Recommandations de l'Académie nationale de médecine, 3/14/2006." 17 pp. www.academie-medecine.fr/sites_thematiques/EOLIENNES/chouard_rapp_14mars_2006.htm.

steel tower. The nacelle is rotated mechanically to face the blades into the wind. The blades spin upwind of the tower. The tower is anchored in a steel-reinforced concrete foundation. Turbine heights in this study were 100 to 135 m (328 to 443 ft) with hub heights 59 to 90 m (194 to 295 ft) and blade lengths 33 to 45 m (108 to 148 ft). Individual turbine powers were 1.5 to 3 MW. Clusters contained from 8 to 45 individual turbines (see Table 1B).

In this study, participants from all families described good and bad symptomatic periods correlated with particular sounds from the turbine installations, rate of turbine spin, or whether the turbines were turned towards, away from, or sideways relative to their homes. All participants identified wind directions and intensities that exacerbated their problems and others that brought relief. Many subjects described a quality of invasiveness in wind turbine noise, more disturbing than other noises like trains. Some stated that the noise wouldn't sound loud to people who did not live with it, or that noises described with benign-sounding terms like "swish" or "hum" were in reality very disturbing. Several were disturbed specifically by shadow flicker, which is the flashing of light in a room as the slanting sun shines through moving turbine blades, or the repetitive movement of the shadows across yards and walls. (These observations are documented in the narrative data of the Case Histories.)

Wind turbines generate sound across the spectrum from the infrasonic to the ultrasonic,[22] and also produce ground-borne or seismic vibration.[23] "In the broadest sense, a sound wave is any disturbance that is propagated in an elastic medium, which may be

---

[22] van den Berg 2004a.

[23] Styles P, Stimpson I, Toon S, England R, and Wright M. 2005. Microseismic and infrasound monitoring of low frequency noise and vibrations from wind farms: recommendations on the siting of wind farms in the vicinity of Eskdalemuir, Scotland. 125 pp. www.esci.keele.ac.uk/geophysics/News/windfarm_monitoring.html

636

a gas, a liquid, or a solid. Ultrasonic, sonic, and infrasonic waves are included in this definition. . . . Sonic waves [are] those waves that can be perceived by the hearing sense of the human being. Noise is defined as any perceived sound that is objectionable to a human being."[24]

Following standard usage in noise literature, I use the word *vibration* to refer to disturbances in solid media, such as the ground, house structures, or the human body. When air-borne sound waves of particular energy (power) and frequency meet a solid object, they may set the object vibrating. Conversely, a vibrating solid object, such as the strings on a violin, can create sound waves in air. There is energy transfer in both directions between air-borne or fluid-borne sound waves and the vibration of solids. When I talk about noise and vibration together, I am referring to this continuum of mechanical energy in the air and solids.

Energy in either form (sound or vibration) can impinge on the human body, and there may be multiple exchanges between air and solids in the path between a source and a human. The tissues of humans and other animals are semi-liquid to varying degrees, and have fluid-filled and air-filled spaces within them, as well as solid structures like bones. As an example of such energy transfer, a sound wave in the air, encountering a house, may set up vibrations in the structure of the house. These vibrations, in walls or windows, may set up air pressure (sound) waves in rooms, which can in turn transmit mechanical energy to the tympanic membrane and middle ear, to the airways and lungs, and to body surfaces. Alternatively, vibrations in house structures or the ground may transmit energy directly to the body by solid-to-solid contact and be conducted through the body by bone conduction.

---

[24] Beranek LL. 2006. Basic acoustical quantities: levels and decibels. Chapter 1 in *Noise and Vibration Control and Engineering: Principles and Applications*, ed. Ver IL, Beranek LL, pp. 1–24. John Wiley & Sons, Hoboken, NJ. p. 1.

637

All parts of the body (and indeed all objects) have specific resonance frequencies, meaning that ***particular frequencies or wavelengths of sound will be amplified in that body part.***[25] If the wavelength of a sound or its harmonic matches the dimensions of a room, it may set up standing waves in the room with places where the intersecting, reverberating sound waves reinforce each other. Resonance also occurs inside air-filled body cavities such as the lungs, trachea, pharynx, middle ear, mastoid, and gastrointestinal tract. The elasticity of the walls and density of the contents of these spaces affect the dynamics of sound waves inside them. The orbits (bones surrounding the eyes) and cranial vault (braincase) are also resonance chambers, because of the lower density of their contents compared to the bones that surround them. There are also vibratory resonance patterns along the spine (which is elastic), including a resonance involving the movement of the head relative to the shoulders. Von Gierke[26,27] and Rasmussen[28] have described the resonant frequencies of different parts of the human body.

Noise intensity is measured in decibels (dB), a logarithmic scale of sound pressure amplitude. Single noise measurements or integrated measurements over time combine the energies of a range of frequencies into a single number, as defined by the filter or weighting network used during the measurement. The A-weighting

---

[25] Hedge, Alan. 2007. Department of Design and Environmental Analysis, Cornell University. Syllabus/lecture notes for DEA 350: Whole-body vibration (January), found at http://ergo.human.cornell.edu/studentdownloads/DEA325pdfs/Human%20Vibration.pdf

[26] von Gierke HE, Parker DE. 1994. Differences in otolith and abdominal viscera graviceptor dynamics: implications for motion sickness and perceived body position. Aviat Space Environ Med 65(8): 747–51.

[27] von Gierke HE. 1971. Biodynamic models and their applications. J Acoust Soc Am 50(6): 1397–413.

[28] Rasmussen G. 1982. Human body vibration exposure and its measurement. Bruel and Kjaer Technical Paper No. 1, Naerum, Denmark. Abstract: Rasmussen G. 1983. Human body vibration exposure and its measurement. J Acoust Soc Am 73(6): 2229.

638

network is the most common in studies of community noise. It is designed to duplicate the frequency response of human hearing for air-borne sounds entering through the outer and middle ear. A-weighting slightly augments the contributions of sounds in the 1000 to 6000 Hz range (from C two octaves above middle C, key 64 on the piano, to F# above the highest note on the piano), and progressively reduces the contributions of lower frequencies below about 800 Hz (G-G# 1½ octaves above middle C, keys 59–60). At 100 Hz, where the human inner-ear vestibular organ has a peak response to vibration[29] (G-G# 1½ octaves below middle C, keys 23–24), A-weighting reduces sound measurement by a factor of 1000 (30 dB). At 31 Hz (B, the second-to-bottom white key, key 3), A-weighting reduces sound measurement by a factor of 10,000 (40 dB). Thus A-weighting preferentially captures the high sounds used in language recognition, to which the human cochlea and outer and middle ear are indeed very sensitive, but reduces the contribution of mid- and lower-range audible sounds, as well as infrasound (defined as 20 Hz and below).

Linear (lin) measurements use no weighting network, so the frequency responses are limited by other aspects of the system, such as microphone sensitivity. Linear measurements may capture low frequency sounds but are not standardized—different sound level meters yield different results. As a result, the standardized and commonly available C-weighting network is preferred for measuring environmental noise with low frequency components, such as noise from wind turbines. The C-weighting network has a flat response (meaning that it does not reduce or enhance the contributions of different frequencies) over the audible frequency range and a well-defined decreasing response below 31 Hz.

---

[29] Todd NPMc, Rosengren SM, Colebatch JG. 2008. Tuning and sensitivity of the human vestibular system to low-frequency vibration. Neurosci Lett 444: 36–41.

One third (1/3) octave band studies are used to describe sound pressure levels by frequency, and are presented as a graph rather than a single number. One third (1/3) octave bands can also be measured linearly or with weighting networks.

Methods

The study design is a case series of affected families, interviewed by telephone. I used a broad-based, structured interview including a narrative account, symptom checklist, past medical and psychiatric history, personal and social history, selected elements of family history, and review of systems. This is the "history" in the standard physician's "history and physical," with specific questions oriented towards the problems in question. The core of the syndrome consists of symptoms such as sleep disturbance, headache, tinnitus, dizziness, nausea, anxiety, concentration problems, and others which are typically diagnosed by medical history more than physical exam.

Limited medical records were provided by the adults of families A and B (A1, A2, B1, B2) and by a young man in family C (C4). I requested records for all families through F, but since no more were forthcoming, I stopped asking, and pursued those parts of the study not dependent on physical examination or test results, and for which I had a uniform study tool, the interview.

The study design includes comparison groups in two ways: 1) I obtained information for each symptom before exposure, during exposure, and away from or after the end of exposure, so that each subject acted as his or her own control in the "natural experiment" of living in a home under a certain set of conditions, having wind turbines added to those conditions, and then moving or going away and again experiencing an environment without turbines. Subjects also noted how their symptom intensity varied in concert with the type and loudness of noise, the direction turbine blades were turned, the rate of spin, or the presence or absence of shadow flicker. A positive symptom is one that emerged from the within-subject comparison as distinctly worse during exposure than before or after (generally both). For example, a subject was considered to have headaches due to turbine exposure only if his (her) headaches were more frequent, severe, or longer-lasting during turbine exposure than his own headaches before being exposed to turbines and after ending the exposure. 2) I obtained information on all household members, not only the most affected, so that I could compare more affected to less affected subjects, all of whom were exposed, to evaluate individual risk factors with regard to age, sex, and underlying health conditions.

Families were selected to conform to all of the following: 1) severity of symptoms of at least one family member; 2) presence of a "post-exposure" condition, in which the family had either left the affected home or spent periods of time away; 3) quality of observation, memory, and expression, so that interviewed people were able to state clearly, consistently, and in detail what had happened to them under what conditions and at what time (all but one individual were native English speakers); 4) residence near recently erected turbines (placed in operation 2004–2007); 5) short time span between moving out and the interview, if exposure had already ended (six weeks was the maximum); and 6) family actions in response to turbine noise showing how serious and debilitating the symptoms were (moving out, purchasing a second home, leaving home for months, renovating house, sleeping in root cellar).

Most families who met these criteria and were willing to be interviewed lived outside the United States. In the course of the study, I received direct evidence that participation by Americans was limited by non-medical factors such as turbine leases or neighbor contracts prohibiting criticism, court decisions restricting criticism of turbine projects, and community relationships. The same factors are likely, in future, to affect other studies of wind turbine noise effects in the United States, with the potential to introduce significant bias into any population-based study.

Moving is an economic hardship for all the families in the study. All own (or owned) their homes, but only three of the eight families who have left their homes have sold them: one to the utility operating the turbines, one to a buyer introduced to the family by the turbine owner, and one to an independent buyer. Three families do not have their homes for sale because the properties include farmland which they farm or lease out. These families have rented additional houses in nearby villages for living and sleeping, though they can ill afford it. The remaining two families who have left their homes are trying to sell the homes, but have not been successful. One of the two families that have not moved is trying to sell their home so they can move. The tenth family has not moved and is not at this point trying to sell the home.

Though not by design, each case household consisted of a married couple or a married couple with children. One family included an older parent. I interviewed both members of each couple except for one man with dementia, and I interviewed the older parent together with her daughter-in-law. I directly interviewed three out of the four subjects in the 16- to 21-year-old age group; the fourth did not make himself available. Child data are otherwise derived from the parent interviews.

I audio-recorded the interviews for the first two families (C and D, in 2006) as I was developing the interview protocol, but after that noted answers directly on an interview form, writing down distinctive or critical observations and symptom descriptions verbatim. Because of subject time constraints, I also audio-recorded the final family (J, in 2008). Subjects who were recorded

642

gave their permission verbally at the beginning of the interview. I made a confidentiality statement and informed subjects that they would have the opportunity to review the data presented about them prior to publication. Follow-up interviews were done with families C, D, and G. Other families have kept in touch by email and telephone about further developments. All ten families have reviewed the information presented about them and signed permission for anonymous publication.

I use simple statistical tests (2x2 χ^2) to examine associations among symptoms and between pre-existing conditions and symptoms during exposure.[30] Degrees of freedom (df) are 2 for all the χ^2 results in this report. Children were excluded from the analysis of adult symptoms if no child younger than a certain age had the symptom in question. Study children were categorized into developmental-age blocks (see Table 1C). When I excluded children from an analysis, I excluded all the children in that age block and below. Excluding children from adult symptom analyses avoided inflating the no symptom/absent pre-existing condition box of the 2x2 χ^2 contingency tables, which could artificially increase the χ^2 value.

Results

I interviewed 23 adult and teenage members of 10 families, collecting information on all 38 adult, teen, and child family members. One family member was a baby born a few days before the family (A) moved out, so there are no data for this child on sleep or behavior during exposure (which was in utero). Thus the sample size of subjects for whom we have information about experiences or behavior during exposure is 37.

[30] Sokal RR, Rohlf FJ. 1969. *Biometry*. W. H. Freeman, San Francisco.

643

Residence status and family composition are detailed in Table 1A; turbine, terrain, and house characteristics in Table 1B; and the age and sex distribution of subjects in Table 1C. Twenty subjects were male and 18 female, ranging in age from <1 to 75. Seventeen subjects were age 21 and below, and 21 subjects were age 32 and above. There is a gap in the 20's and a preponderance of subjects in their 50's. Wind turbine brands to which study subjects were exposed included Gamesa, General Electric, Repower, Bonus (Siemens), and Vestas.

Individual accounts of baseline health status and pre-exposure, during exposure, and post-exposure symptoms or absence of symptoms are presented in the CASE HISTORIES for families A through J, with a separate sub-table (A1, A2, A3, etc.) for each individual. I encourage the reader to read these, because they highlight the before-during-after comparisons for each person, show how the symptoms fit together for individuals, reveal family patterns, and provide subjects' own words for what they feel and detect. When individuals are referred to in the text, the letter and number in parentheses (e.g., A1, C2) refers to the CASE HISTORY table in which that subject's information is found.

Baseline conditions

Eight adult subjects had current or history of serious medical illness, including lupus (1), breast cancer (2), diabetes (1), coronary artery disease (2), hypertension (1), atrial fibrillation with anticoagulation (1), Parkinson's disease (1), ulcer (1), and fibromyalgia (2). Two were male (age 56–64) and six female (age 51–75). Other past and current medical illnesses are listed in Table 2. Four subjects smoked at the beginning of exposure, and five others had smoked in the past (Table 2). There were no seriously ill children in the sample.

Seven subjects had histories of mental health disorders including depression, anxiety, post-traumatic stress disorder (PTSD), and

644

bipolar disorder. Three were male (age 42–56) and four female (age 32–64). One of these men (age 56) also had Alzheimer's disease. There were no children with mental health disorders or developmental disabilities in this sample.

Eight subjects had pre-existing migraine disorder (including two with previous severe sporadic headaches that I interpreted as migraine). Four were male (age 19–42) and four female (age 12–42). An additional seven subjects, age <1 to 17, were children of migraineurs who had not experienced migraines themselves at baseline.

Eight subjects had permanent hearing impairments, defined subjectively or objectively, including mild losses, losses limited to one ear, or impairments of binaural processing. Six were male (age 32–64) and two female (age 51–57).

Six subjects had continuous tinnitus or a history of multiple, discrete episodes of tinnitus prior to exposure. Four were male (age 19–64) and two female (age 33–57).

Twelve subjects had significant previous noise exposure, defined as working in noisy industrial or construction settings; working on or in a diesel boat, truck, bus, farm equipment, or aircraft; a military tour of duty; or operating lawn mowers and chain saws for work. Not included were home or sporadic use of lawn mowers and chain saws, commuting by train or airplane, urban living in general, or playing or listening to music. Nine of the noise-exposed subjects were male (age 19–64) and three female (age 33–53).

Eighteen subjects were known to be motion sensitive prior to exposure, as defined by carsickness as a child or adult, any episode of seasickness, or a history of two or more episodes of vertigo. Ten were male (age 6–64) and eight female (age 12–57).

Case	Country	# in household	# interviewed	Ages†	Head of household occupations	Residence status
F	UK	4	4	51, 42, 17, 75**	Farmer, nurse midwife	Rented house in nearby village and continue to use farm and home office during day.
G	Ireland	6	2	35, 32, 6, 5, 2, 8 months	Computer programmer, homemaker	Under pressure from family, turbine owner arranged purchase at 30% below pre-turbine value.
H	Ireland	3	2	57, 52, 8	Milk truck driver, homemaker	Family occupies home. Significant renovations made in attempt to exclude noise.
I	Italy	2	2	59, 52	Professional gardener, teacher	Occupied newly built home during study but wife spent months away due to symptoms. Moved out after study completed, leaving home vacant and for sale.
J	USA	4	2	49, 47, 13, 8	Physician, nurse	Family occupies home.

*Families A and B are related and own separate homes on the same property.
**Grandmother living in different house on same property did not move away.
†Underlined ages indicate interviewees.

645

Table 1B: Cases: physical attributes

Case	Distance to closest turbine	# turbines	MW per turbine	Year placed in operation	Hub height	Total height	Terrain	Configuration of turbines	House construction
A	1000 m (3281 ft)	10	3	2007	90 m	135 m	Hilly with rocky ridges	10 in line point at house at hub level	Wood frame
B*	1000 m (3281 ft)	10	3	2007	90 m	135 m	Hilly with rocky ridges	10 in line point at house at hub level	Wood frame
C	305 m (1000 ft)	17	1.8	2004–05	80 m	125 m	Rocky peninsula	On three sides	Wood frame
D	548 m (1798 ft)	22	1.8	2006	78 m	117 m	Flat farmland	Group on one side	Wood frame
E	423 m (1388 ft)	45	1.5	2006	87 m	120 m	Flat farmland, swamp	On three sides	Brick with stone front
F	930 m (3051 ft)	8	2	2006	59 m	100 m	Flat farmland	5 in line point at house	Brick on cement slab
G	596 m (1955 ft)	32	3	2006	80 m	125 m	Rocky hills	Above house on three sides	Stone cottage, walls 60 cm thick
H	1500 m (4921 ft)	11	2.3	2005	80 m	121 m	Rocky hills	Above house on three sides	Stone cottage, cement slab
I	875 m (2871 ft)	10	2	2006	78 m	121 m	Rocky hills	Across valley at higher elevation	Stone and brick, walls 50 cm thick
J	732 m (2400 ft)	40	2	2007	80 m	123 m	Ridges and valleys	6 in L-shape above house on two sides	Wood frame

*Families A and B are related and own separate homes on the same property.

Table 1A: Cases: personal attributes

Case	Country	# in household	# interviewed	Ages†	Head of household occupations	Residence status
A	Canada	4	2	33, 32, 2½, 2 months	Fisherman, accountant	Moved to a rented house in nearby village 6 months after renovating their own home, which is vacant. Land they own continues to be leased to a farmer.
B*	Canada	3	3	55, 53, 19	Fisherman, homemaker	Moved to a rented house in nearby village. Home is vacant. Land they own, which has been in the family for over a century, continues to be leased to a farmer.
C	Canada	8	3	45, 42, 21, 19, 15, 12, 9, 5	Fisherman, homemaker	Family divided and moved in with extended family members. Home, built 24 years before by husband on land in family for over a century, is vacant and for sale.
D	Canada	2	2	64, 64	Retired/disabled, home health aide	Occupied home, purchased second house in village 25 miles away during study. Sold home and moved after study completed.
E	Canada	2	1	56, 56	Retired/disabled, teacher	Moved to a newly purchased house in a nearby village after turbine utility bought their home and property.

647

648

Table 1C: Cases: demographics

Age	Male	Female	Total
<1	1	1	2
1–3	1	1	2
4–6	2	1	3
7–11	3	0	3
12–15	1	2	3
16–21	2	2	4
22–29	0	0	0
30–39	2	2	4
40–49	3	2	5
50–59	4	5	9
60–69	1	1	2
70–79	0	1	1
Totals	20	18	38

649

The subjects' baseline conditions are summarized in Table 3.

Seven subjects had a remembered history of a single concussion, and none had a history of a more severe head injury. Six were male (age 19–59) and one female (age 12). I did not collect information on whiplash injury.

Core symptoms

Core symptoms are defined as 1) common and widely described by study participants, 2) closely linked in time and space to turbine exposure, and 3) amenable to diagnosis by medical history. Core symptoms include sleep disturbance, headache, tinnitus, other ear and hearing sensations, disturbances to balance and equilibrium, nausea, anxiety, irritability, energy loss, motivation loss, and disturbances to memory and concentration.

An additional core symptom is a new type of internal or visceral sensation which has no name in the medical lexicon. Subjects struggled to explain these sensations, often apologizing for how strange their words sounded. A physician subject called it "feeling jittery inside" or "internal quivering." Other subjects chose similar words, while others talked about feeling pulsation or beating inside. The physical sensations of quivering, jitteriness, or pulsation are accompanied by acute anxiety, fearfulness, or agitation, irritability, sleep disturbance (since the symptom arises during sleep or wakefulness), and episodes of tachycardia. I call this sensation and accompanying symptoms *Visceral Vibratory Vestibular Disturbance* (VVVD). It is described further below.

Core symptoms are closely correlated with exposure, including being at home, the direction and strength of the wind, whether turbines are facing the home, and the presence of moving blade shadows. Core symptoms all resolve immediately or within hours away from the turbines, with the exception of disturbances of

650

concentration and memory, which resolved immediately in some cases or improved over weeks to months in others.

Core symptoms are summarized in Table 3.

Sleep disturbance. Thirty-two subjects (17 males age 2–64 and 15 females age 2–75) had disturbed sleep. Types of sleep disturbance included: difficulty getting to sleep, frequent or prolonged awakening by turbine noise, frequent or prolonged awakening by awakened children, night terrors (both 2½-year-olds, B3 and G5), nocturnal enuresis (one 5-year-old girl, G4), nocturia (six women age 42–75 and one man age 64; B2, C2, E2, F2, F4, H2, D1), excessive movement during sleep (one 8-year-old boy, H3), excessive nighttime fears (two 5-year-olds, a girl and a boy, C8 and G4), and abrupt arousals from sleep in states of fear and alarm (four women age 42–57; C2, F2, H2, I2). Other adults, though not fearful when they woke up, awoke with physical symptoms similar to their daytime symptoms of anxiety/agitation/internal quivering (three men age 42–64 and two women age 32–53; D1, F1, J1, B2, G2). Four people slept well, including the one infant (G6), a 19-year-old woman (B3), a 47-year-old woman (J2) and her 8-year-old son (J4). It was unclear whether a 56-year-old man with dementia, bipolar disorder, Parkinson's disease, and disturbed sleep at baseline (E1) slept worse than usual or not.

With three exceptions, all types of sleep disturbance resolved immediately whenever subjects slept away from their turbine-exposed homes, including the adult nocturia and the 5-year-old's nocturnal enuresis. A 49-year-old man with a pre-existing sleep disturbance (J1) took two nights to get back to his baseline, and a 45-year-old man (C1) and a 42-year-old man (F1) did not improve all the way to baseline; this was thought to be due to coexisting depression after abandoning their homes.

651

Table 2: Past and current serious medical illness

	Adult (>22 yo) (n=21)		Child/youth (0–21 yo) (n=17)	
	Male	Female	Male	Female
Breast cancer		2		
Skin cancer	1			
Lupus		1		
Diabetes	1			
Polycystic ovarian syndrome		1		
Coronary artery disease	1	1		
Atrial fibrillation with anticoagulation		1		
Other arrhythmias	1	1		
Hypertension—present		1		
Hypertension—past or pregnancy		2		
Parkinson's disease	1			
Diplopia		1		
Renal function impairment	1			
Ulcer—past	1			
Gastroesophageal reflux	2	3		
Irritable bowel syndrome	1	1		
Fibromyalgia		2		
Osteoarthritis	1	1		
Back pain	2	1	1	
Other joint pain	1			
Asthma	2	2		1
Eczema		1	1	
Frequent/chronic otitis media—present			1	1
Frequent/chronic otitis media—past		1	2	1
Smoking—present	3	1		
Smoking—past	3	1	1	

652

Table 3: Baseline conditions and core symptom occurrence*

	Total	Male	Ages	Female	Ages	N**	% of sample
Baseline Conditions							
Serious medical illness†	8	2	56–64	6	51–75	38	21
Mental health disorders‡	7	3	42–56	4	32–64	34	21
Migraine disorder	8	4	19–42	4	12–42	34	24
Hearing impairments	8	6	32–64	2	51–57	34	24
Pre-existing tinnitus	6	4	19–64	2	33–57	24	25
Previous noise exposure	12	9	19–64	3	33–53	24	38
Motion sensitivity	18	10	6–64	8	12–57	34	53
Core Symptoms							
Sleep disturbance	32	17	2–64	15	2–75	36	89
Headache	19	8	6–55	11	12–57	34	56
VVVD◊	14	6	32–64	8	32–75	21	67
Dizziness, vertigo, unsteadiness	16	7	19–64	9	12–64	27	59
Tinnitus	14	9	19–64	5	33–57	24	58
Ear pressure or pain	11	6	2–25	5	19–57	36	30
External auditory canal sensation	5	2	42–55	3	52–75	34	15
Memory and concentration deficits (salient+mild/vague)	28	15	6–64	13	5–57	30	93
Irritability, anger	28	15	2–64	13	2–64	37	76
Fatigue, loss of motivation	27	14	2–64	13	2–75	36	75

*A symptom during exposure is defined as distinctly worse for that individual during exposure compared to before and/or after exposure.
**N=number of subjects in which it was possible to know about the condition or symptom, given age and other specific limitations (see p. 41 and subsequent text).
†See p. 42 and Table 2.
‡See p. 42 and subsequent text for definitions of this and other conditions and symptoms.
◊Visceral Vibratory Vestibular Disturbance: See pp. 48 and 55ff.

Headache. Nineteen subjects experienced headaches that were increased in frequency, intensity, and/or duration compared to baseline for that person. Eight were male (age 6–55) and eleven female (age 12–57). Eight had pre-existing migraine (C2, C3, C4, C5, C6, F1, G1, G2). Two women (one a migraineur, one not; C2, E2) had severe headaches provoked by shadow flicker. All other exposure-related headaches were triggered by noise alone. Recovery from headaches generally took several hours after the exposure ended.

Headache risk factors were examined in a subset of the study group that included all subjects age 5 and older (N=34), since the younger children in the study (age <1 to 2) were not reliable sources of information on headache. The occurrence of unusually severe or frequent headaches during exposure was significantly associated with pre-existing migraine disorder ($\chi^2 = 8.26$, $p = 0.004$). All 8 subjects with pre-existing migraine experienced headaches that were unusually intense, frequent, or prolonged compared to their baseline headaches. Of the 26 subjects without pre-existing migraine, 11 also experienced unusual or severe headaches during exposure. Two of these were children of migraineurs not known to have migraine themselves (a girl age 17 and a boy age 6; F3, G3). All children or teens (through age 21) who had headaches during exposure were migraineurs or children of migraineurs.

Once migraine was factored out as a risk factor, 9 of 17 subjects over age 22 without a history of migraine still had headaches of increased intensity, duration, or frequency during exposure to turbines. I found no significant correlation within this group between headache and the presence of serious underlying medical illness ($\chi^2 = 0.486$, $p = 0.486$), present or past mental health disorder ($\chi^2 = 0.476$, $p = 0.490$), tinnitus or hearing loss at baseline, motion sensitivity at baseline, or tinnitus, disequilibrium, or VVVD during exposure.

In summary, a little more than half (19) of the 34 study participants age 5 and older experienced unusually severe headaches during exposure. Migraine was a statistically significant risk factor but was present in fewer than half (8) of the 19 subjects with worsened headache. Children and teens up to age 21 with headaches either had known migraine or were the children of migraineurs. Nine of the 19 headache subjects were adults without clear risk factors, showing that while people with migraine are more likely to have headaches of unusual intensity, duration, or frequency around turbines, so can other adults without identified risk factors.

Ears, hearing, and tinnitus. Fourteen subjects (nine males age 19–64 and five females age 33–57) experienced tinnitus that was new or worse in severity or duration than at baseline. For two men (age 55 and 64; B1, D1), the tinnitus at times interfered with their ability to understand conversation. Four of the 14 subjects experienced particularly disturbing kinds of tinnitus or noise which was perceived to be inside the head (two men age 42, 55, and two women age 52, 57; B1, F1, H2, I2). This sensation was painful for two subjects. Tinnitus tended to resolve over several hours after exposure ended.

Tinnitus risk factors were examined in subjects age 16 and older, since the youngest person with tinnitus was in this age group. The subject with dementia (E1) was excluded, since there was no information on his hearing status or tinnitus. Sample size was 24 subjects. The occurrence of new or worsened tinnitus in the presence of turbines was significantly correlated with previous noise exposure ($\chi^2 = 6.17$, $p = 0.013$), tinnitus prior to exposure ($\chi^2 = 5.71$, $p = 0.017$), and baseline hearing loss ($\chi^2 = 4.20$, $p = 0.040$). New or worsened tinnitus during exposure was strongly correlated with ear popping, ear pressure, or ear pain during exposure ($\chi^2 = 7.11$, $p = 0.008$), and weakly correlated with dizziness/ disequilibrium during exposure ($\chi^2 = 3.70$, $p = 0.054$). Tinnitus during exposure did not show a significant relationship with pre-existing migraine or motion sensitivity, or with headache or VVVD during exposure.

Eleven subjects during exposure experienced ear popping, ear or mastoid area pressure, ear pain without infection, or a sensation that the eardrum was moving but not producing a sensation of sound (six males age 2–55 and five females age 19–57). The 2½-year-old (A3) pulled on his ears and got cranky repeatedly at the same time as his grandmother's (B2) exacerbations of headache, tinnitus, and ear pain. Correlations with tinnitus during exposure are described above. Five subjects experienced tickling, blowing, or undefined sensations in the external auditory canal, or increased wax production (two men age 42, 55, and three women age 52–75).

Individual subjects noticed changes in their hearing or auditory processing. A 33-year-old woman (A2) had progressively worsening tinnitus during her five months of exposure. After she moved away, the tinnitus resolved and she noticed she had a new difficulty understanding conversation in a noisy room, now needing to watch the speaker's face carefully. Her son (A3, the 2½-year-old who pulled on his ears and got cranky, above) did not confuse sounds before exposure, but began to do so during exposure, and continued to do so at the time I interviewed his mother six weeks after the exposure ended. The child's language development was otherwise good. A 42-year-old woman (C2) had tinnitus throughout her 21-month exposure period without subjective hearing changes. After she moved and the tinnitus resolved, she noted hyperacusis. A 32-year-old woman (G2) experienced hyperacusis during exposure, but no tinnitus. The hyperacusis resolved after the family moved.

Balance and equilibrium. Sixteen subjects (seven males age 19–64 and nine females age 12–64) experienced disturbance to their balance or sense of equilibrium during exposure, describing

656

dizziness, light-headedness, unsteadiness, or spinning sensations. One of them, a 42-year-old woman (C2), described how a friend, sitting next to her in her turbine-exposed home, remarked how her (C2's) eyes appeared to be bouncing back and forth (nystagmus). Ten of these 16 subjects also experienced nausea during exposure to turbines, during or separate from dizziness. No children under the age of 12 had symptoms of dizziness, disequilibrium, or nausea during exposure, except for the usual nausea of acute gastrointestinal and other infections.

Risk factors for dizziness/disequilibrium in the presence of turbines were analyzed using subjects age 12 and up, since this was the youngest age child with this type of symptom. The subject with Parkinson's disease and dementia (E1) was excluded because his baseline balance problems and inability to express himself made it hard for his wife (the informant) to tell if he had worsened symptoms during exposure or not. The remaining sample was 27 subjects. Disequilibrium during exposure was significantly correlated with headaches during exposure ($\chi^2 = 5.08$, $p = 0.024$) and baseline motion sensitivity ($\chi^2 = 4.20$, $p = 0.040$). Disequilibrium during exposure is weakly correlated with tinnitus during exposure ($\chi^2 = 3.70$, $p = 0.054$). (Inspection of the data shows that these are primarily ataxic (unsteady) subjects.) Dizziness/disequilibrium during exposure was not correlated with VVVD or ear popping/pressure/pain during exposure, pre-existing migraine disorder, previous noise exposure, or prior tinnitus or hearing loss.

Internal quivering, vibration, or pulsation. Eleven adult subjects described these uncomfortable, unfamiliar, and hard-to-explain sensations:

- Dr. J (J1, age 49) described "internal quivering" as part of the "jittery feeling" he has when the turbines are turning fast.

657

- Mrs. I (I2, age 52) said the noise inside her house is "low, pulsating, almost a vibration," not shut out by earplugs. She gets a sensation inside her chest like "pins and needles" and chest tightness on awakening at night to noise. "It affects my body—this is the feeling I get when I say I'm agitated or jittery. It's this that gives me pressure or ringing in my ears." "A feeling someone has invaded not only my health and my territory, but my body."
- Mrs. H (H2, age 57) described a pulsation that prevented sleep from the "unnatural" noise from the turbines.
- Mr. G (G1, age 35) described feeling disoriented and "very strange" in certain parts of the house where he could "feel rumbling." If he did not move quickly away from these locations, the feeling would progress to nausea. He described the noise as "at times very invasive. Train noise has a different quality, and is not invasive."
- Mrs. G (G2, age 32) felt disoriented, "light-headed," dizzy, and nauseated in her garden and in specific parts of the house where she detected vibration. She felt her body vibrating "inside," but when she put her hand on walls, windows, or objects, they did not seem to be vibrating.
- Mrs. F (F2, age 51) described a physical sensation of noise "like a heavy rock concert," saying the "hum makes you feel sick."
- Mrs. E (E2, age 56), when supine, felt a "ticking" or "pulsing" in her chest in rhythm with the audible swish of the turbine blades. She interpreted this as her "heart synchronized to the rhythm of the blades," but there is no information (such as a pulse rate from the wrist at the same time) to determine whether this was true or not, or whether she detected a separate type of pulsation. Mrs. E could make these sensations go away by getting up and moving around, but they started again when she lay back down.

658

- Mr. D (D1, age 64) felt pulsations when he lay down in bed. In addition, "When the turbines get into a particular position (facing me), I get real nervous, almost like tremors going through your body... it's more like a vibration from outside... your whole body feels it, as if something was vibrating me, like sitting in a vibrating chair but my body's not moving." This occurs day or night, but not if the turbines are facing "off to the side."
- Mr. C (C1, age 45) felt pulsations in his chest that would induce him to hold his breath, fight the sensation in his chest, and not breathe "naturally." Chest pulsations interrupted his sleep and ability to read. He also described a sensation of "energy coming within me... like being cooked alive in a microwave."
- Mrs. B (B2, age 53) described her breath being "short every once in a while, like [while] falling asleep, my breathing wanted to catch up with something."
- Mr. B (B1, age 55) had two episodes of feeling weight on his chest while lying down, which resolved when he stood up. Other than this, he experienced the invasive quality of the noise in his head and ears: "That stuff [turbine noise] doesn't get out of your head, it gets in there and just sits there—it's horrible."

Agitation, anxiety, alarm, irritability, nausea, tachycardia, and sleep disturbance are associated with internal vibration or pulsation:

- Dr. J's (J1, age 49) "jittery" feeling includes being "real anxious," irritable, and "no fun to be around." He interrupts outdoor and family activities to sequester himself in his well-insulated house. When the turbine blades are spinning fast and he detects certain types of noise and vibration as he arrives home from work, he gets queasy and loses his appetite. He awakens from sleep with the "jittery" feeling and tachycardia, and may need to go downstairs to a cot in the 55-degree root cellar (the

659

only place on his property where he cannot hear or feel the turbines) to be able to fall back to sleep. He often takes deep breaths or sighs when in the "jittery" state.

- Mrs. I (I2, age 52) describes episodic "queasiness and nausea" with loss of appetite, "trembling in arms, legs, fingers," "strong mental and physical agitation," and frequent unexpected crying. On noisy nights she awakens after four hours of sleep, weeping in the night. "When I wake up, [there is] more a feeling of pressure and tightness in my chest; it makes me panic and feel afraid." It is "a startling sort of waking up, a feeling there was something and I don't know what it was." Once she awoke thinking there had been an earth tremor (there had not), and twice she has awakened with tachycardia, the "feeling your heart is beating very fast and very loud, so I can feel the blood pumping." Feelings of panic keep her from going back to sleep.
- Mrs. H (H2, age 57) awakens 5–6 times per night with a feeling of fear and a compulsion to check the house. She describes it as a "very disturbed sort of waking up, you jolt awake, like someone has broken a pane of glass to get into the house. You know what it is but you've got to check it—go open the front door—it's horrific." She finds it hard to fall back to sleep and describes herself as irritable and angry, shouting more at her family members.
- Mr. G (G1, age 35) described the noise outside his home and the noise that awakened him at night as "stressful."
- Mrs. G (G2, age 32) was, during exposure, irritable, angry, and worried about the future and her children. She awoke often at night because her children woke up, when she cared for their fears, mentioning none of her own.
- Mrs. F (F2, age 51) described a "feeling of unease all the time." At night she startles awake with heart pounding, a feeling of

66D

fear, and a compulsion to check the house. The feeling of alarm keeps her from being able to go back to sleep.

- Mrs. E (E2, age 56) did not express anxiety or fear, but she awakened repeatedly at night and was unable to get back to sleep on nights when the turbines were facing the house.
- Mr. D (D1, age 64) described how he has to "calm down" from the "tremor." If outside, "I come in, sit down in my chair and try to calm myself down. After an episode like that, I'm real tired." Mood has worsened with increased anger, frustration, and aggression. Tachycardia accompanies the "tremor" at times: "My heart feels like it's starting to race like crazy and I have these tremors going through my body." Mr. D pants or hyperventilates when the tremor and tachycardia occur, and consciously slows his breathing when calming down.
- Mr. C (C1, age 45) was unable to rest, relax, or recuperate in his home, where his body was "always in a state of defense." He had to drive away in his car to rest.
- Mrs. B (B2, age 53) became "upset and in a turmoil" when her symptoms worsened, leaving her house and tasks repeatedly to get relief.
- Mr. B (B1, age 55) described stress, "lots, pretty near more'n I could take, it just burnt me, the noise and run-around." He was prescribed an anxiolytic, and spent more time at the shore in his fishing boat for symptom relief.

The internal quivering, vibration, or pulsation and the associated complex of agitation, anxiety, alarm, irritability, tachycardia, nausea, and sleep disturbance together make up what I refer to as *Visceral Vibratory Vestibular Disturbance* (VVVD). Fourteen adult subjects (six men age 35–64 and eight women age 32–75) had VVVD during exposure, including the eleven quoted above and Mr. F (F1, age 42), Mrs. F Senior (F4, age 75), and Mrs. C (C2, age 42). Mr. I (I1, age 59) had partial symptoms, with an urge to escape, noise-induced nausea, and sleep disturbance, but no feeling of internal movement. VVVD resolves immediately upon leaving the vicinity of the turbines, when the turbines are still and silent, and under favorable weather conditions at each locality.

Because VVVD is in part a panic attack, accompanied by other physical and mental symptoms, I examined the relationships among VVVD and panic disorder, other mental health diagnoses, and other risk factors. The sample for this analysis was 21 adults ages 22 and above (since the study had no participants age 22–29, this is the same for this study as starting with the age group of the youngest symptomatic subjects, who were 32).

No study subjects had pre-existing panic disorder or previous isolated episodes of panic, so there was no correlation between pre-existing panic and VVVD. Seven subjects had mental health disorders either at the time turbines started up near their homes (two subjects) or in the past (five subjects), including depression, anxiety, post-traumatic stress disorder (PTSD), and bipolar disorder. There was no correlation between current or past mental health disorder and VVVD ($\chi^2 = 0.429$, $p = 0.513$). There was, however, a highly significant correlation between VVVD and motion sensitivity ($\chi^2 = 7.88$, $p = 0.005$).

There was also a moderately significant correlation between VVVD and headaches during exposure ($\chi^2 = 4.95$, $p = 0.026$). There was no correlation between VVVD and dizziness or tinnitus during exposure, or between VVVD and pre-existing migraine, tinnitus, or hearing loss.

Concentration and memory. Twenty of the 34 subjects age 4 and up (eleven males age 6–64 and nine females aged 5–56) had salient problems with concentration or memory during exposure

662

to wind turbines, compared to pre- and/or post-exposure. This is a conservative count, including only subjects whose accounts included specific information on decline in school and homework performance (for children and teens) or details on loss of function for adults. Eight other subjects had some disturbance to concentration and memory, but symptoms were milder or the descriptions more vague (in their own or parents' accounts). Five other subjects, all older adults, noted no change compared to pre-existing memory problems. This leaves only one subject, a 19-year-old woman home from college and minimally exposed (B3), who did not have baseline deficits and was unaffected.

Pre-exposure cognitive, educational, and work accomplishments, specific difficulties related to concentration and memory during exposure, and degree and timing of post-exposure recovery are documented in the CASE HISTORIES for each individual, under "Cognition." Difficulties are often striking compared to the subject's usual state of functioning:

- Mr. A (A1, age 32), a professional fisherman with his own boat, who had an isolated difficulty with memory for names and faces prior to exposure, became routinely unable to remember what he meant to get when he arrived at a store, unless he had written it down.
- Mrs. B (B2, age 53), a homemaker, got confused when she went to town for errands unless she had written down what she was going to do, and had to return home to get her list. When interviewed six weeks after moving, she reported that she had improved to being able to manage three things to do without a list.
- Mr. C (C1, age 45) had to put reading aside because he could not concentrate whenever he felt pulsations.

663

- Mrs. C (C2, age 42), a very organized mother of six who was "ready a month in advance for birthday parties" prior to exposure, became disorganized and had difficulty tracking multiple tasks at once, including while cooking, repeatedly boiling the water away from pots on the stove. She remarked, "I thought I was half losing my mind."
- Mr. D (D1, age 64), a disabled, retired industrial engineer, noticed progressive slowing of memory recall speed and more difficulty remembering what he had read.
- Mrs. E (E2, age 56), a retired teacher active in community affairs, could not spell, write emails, or keep her train of thought on the telephone when the turbine blades were turned towards the house, but was able to do these things when the blades were not facing the house.
- Mrs. F (F2, age 51), a nurse, child development specialist, midwife, and master's level health administrator, could not follow recipes, the plots of TV shows, or furniture assembly instructions during exposure.
- Mrs. G (G2, age 32), a well-organized mother of four, was forgetful, had to write everything down, could not concentrate, and could not get organized. She forgot a child's hearing test appointment. She did not have memory or concentration problems during a previous depression at age 18, and described her experience as "different this time.".
- Mr. I (I1, age 59), a professional gardener, could not concentrate on his outdoor gardening and building tasks if the turbines were noisy, saying "after half an hour you have to leave, escape, close the door."
- Dr. J (J1, page 49), a physician, noticed marked concentration problems when he sat down to pay bills in a small home office with a window towards the turbines.

664

Decline in school performance compared to pre-exposure, or marked improvement in school performance after moving away from turbines, was noted for 7 of the 10 study children and teens attending school (age 5–17; C7, F3, G3, G4, H3, J3, J4). For example:

- A 17-year-old girl (F3), a diligent student, was not concerned about the turbines and thought her parents were overdoing their concern until she unexpectedly did worse on national exams than the previous year, surprising her school, family, and self. At this point she began accompanying her parents to their sleeping house.
- A 9-year-old boy (C7), whose schoolwork was satisfactory without need for extra help prior to exposure, failed tests, lost his math skills, and forgot his math facts. He could not maintain his train of thought during homework, losing track of where he was if he looked up from a problem.
- A 6-year-old boy (G3), described as an extremely focused child and advanced in reading prior to exposure, did not like to read during exposure. Two months post-exposure, now age 7, he would sit down to read on his own for an hour at a time, reading "quite a thick book" for his age.
- His 5-year-old sister (G4) had a short attention span prior to exposure. Her hearing loss due to bilateral chronic serous otitis media was thought to be interfering with schoolwork during exposure, and she repeatedly had tantrums over schoolwork at home during the exposure period. Two months after moving, despite no change in her ears (on a waiting list for pressure equalization tubes), she was more patient and could work longer on homework. Her mother noted that her "schoolwork has improved massively."
- An 8-year-old boy (H3) had an excellent memory and did well in reading, spelling, and math prior to exposure. During exposure he became resistant to doing homework, with tantrums, and

his teacher told him he was not concentrating and needed to go to bed earlier.

In comparing the 20 subjects with salient concentration or memory changes to the 14 who had no change from baseline or vague/ minimal difficulties, there are significant relationships with 1) baseline cognition, in that those without memory or concentration deficits at baseline are more likely to notice such deficits during exposure ($\chi^2 = 4.86$, $p = 0.027$), and 2) fatigue or loss of energy or enjoyment for usual activities during exposure ($\chi^2 = 5.61$, $p = 0.018$). There is no significant relationship between salient concentration or memory changes and pre-existing psychiatric diagnoses, migraine, motion sensitivity, or noise exposure, or between salient concentration or memory changes and headache, tinnitus, VVVD, or irritability during exposure.

In addition to the statistical association between fatigue and concentration disturbance, a number of subjects directly attributed their concentration problems to their sleep deprivation or disturbance. Several aspects of the data, however, suggest that additional factors may be involved.

First, one subject, Mrs. E (E2, age 56), could not do certain mental tasks requiring concentration when the turbines were turned towards her house, but could do them when the turbines were not turned towards the house. Mr. C (C1, age 45), Mr. I (I1, age 59), and Dr. J (J1, age 49) also had concentration problems closely linked in time and space to direct exposure to turbine noise.

Second, some of the problems described by subjects, such as Mrs. F (F2, age 51) and the members of families A and B, are more extreme than I expect from sleep deprivation. The degree of thinking dysfunction involved in not being able to follow a recipe or assemble a piece of furniture, in a woman both highly educated and involved in several practical professions (nursing and farming), does not match my expectation of sleep deprivation from the experience, for example, of both younger and older physicians, who often function under sleep deprivation.

Third, some subjects had concentration problems without obvious sleep problems. All four members of family J had concentration problems, but only Dr. J (J1, age 49) was sleep deprived. Mrs. J (J2, age 47) fell asleep easily and usually went back to sleep if awakened, but still had problems with memory and focus in her home activities that she had noticed and attempted to treat. Their 13-year-old son (J3) needed white noise or music to drown out turbine noise to fall asleep, but went to sleep promptly, slept through the night, and did not complain in the morning of being tired or having slept poorly. His school performance and his level of distractibility at home, however, were both markedly different than at baseline. The younger son, age 8 (J4), continued to sleep well, but still had a surprising decline in school performance, though milder and of shorter duration than his brother's.

Fourth, the problems with concentration and memory resolve on a different schedule from the turbine-related sleep problems. Sleep problems resolve immediately except when accompanied by persistent depression (C1, F1). Problems with concentration and memory frequently took longer to improve, even in the absence of depression. To study resolution, we need to look at subjects who have moved away from their exposed homes or spent a prolonged period away that included work (families A, B, C, E, F, and G, and Mrs. I), since vacations do not provide the same challenges to concentration and memory. Of these 23 subjects over age 4, 13 had salient difficulties with concentration or memory:

- Mr. A (A1, age 32) rated his memory as 85% at baseline, 2% during exposure, and 10% six weeks after moving away.

667

- Mr. and Mrs. B (B1, B2, age 55 and 53) said their memories had partially recovered six weeks after moving.
- Mr. C (C1, now age 47), with continuing depression and ongoing exposure for house maintenance, noted 25 months after moving how bad his memory seemed.
- Mrs. C (C2, now age 44) felt she had recovered her memory and concentration 18 months after moving, despite ongoing stress from crowded living arrangements. Her affected son (now age 11, C7) had not completely recovered his school performance.
- Mrs. E (age 52) recovered immediately. She only experienced problems during exposure when the turbines were turned in a particular direction.
- Mr. and Mrs. F (F1, F2, ages 42 and 51) had moved away but still worked at their turbine-exposed home and farm during the day. Three months after they moved, both thought their concentration had improved, but not to baseline. Mr. F, with ongoing depression, did not perceive any memory recovery. I do not have information about their daughter's (F3, age 17) exam performance after moving.
- Mrs. G (G2, age 32) rated her memory as 10/10 at baseline, 2/10 during exposure, and 5/10 two months after moving away, at which point her depression was mostly resolved. Mrs. G's 5-year-old and 6-year-old children (G3, G4) showed marked improvements in concentration by two months after moving.

Only three subjects were clearly depressed during or after exposure. Mrs. G (G2, age 32) was becoming depressed at the time of the first (during exposure) interview. She remarked on the difference in her cognitive functioning between her current experience and a previous episode of depression at age 18, when she had no problem with her memory or concentration. Two other subjects, Mr. C (C1, age 45) and Mr. F (F1, age 42), developed depression after they had to abandon their homes, which was associated with prolonged memory difficulties. Both also had ongoing exposure.

Irritability and anger. Twenty-eight subjects (15 male age 2–64 and 13 female age 2–64) perceived themselves or were noted by parents to be more angry, irritable, easily frustrated, impatient, rude, defiant, or prone to outbursts or tantrums than at baseline. The adults were uniformly apologetic about their own irritability, and several described how careful they were to avoid acting irritable in their households. Four children (three boys age 8–9 and a girl age 5; C7, G3, H3, G4) were markedly frustrated over homework. The young children of family G quarreled and had tantrums incessantly, and the six children/young adults in family C became angry, prickly, moody, defiant, or prone to fights at school. In families with children, the breakdown in children's behavior, social coping skills, and school performance was one of the strongest elements propelling them to move.

Fatigue and motivation. Twenty-one subjects felt or acted tired, and 24 had problems with motivation for usual, necessary, or formerly enjoyable activities (27 combined, 14 male age 2–64 and 13 female age 2–75). Like concentration and memory, these symptoms undoubtedly have a relationship with sleep deprivation, but certain subjects described leaden feelings around turbines that resolved as soon as they left the vicinity, such as Mr. A (A1, age 32), who said, "You feel different up there: draggy, worn out before you even start anything. . . . It was a chore to walk across the yard." After driving an hour away to visit a family member, "I felt better all over, like you could do a cart wheel," and he felt well after moving.

When away from their turbine-exposed homes, most subjects recovered their baseline positive mood states, energy, and motivation immediately. Six adult subjects did not. These were Mr. B (B1, age 55), Mr. and Mrs. C (C1, C2, age 45 and 42), Mr. and

669

Mrs. F (F1, F2, age 42 and 51), and Mrs. G (G2, age 32). By their own accounts, three (Mr. C, Mr. F, and Mrs. G) had unresolved or resolving depression. All but Mrs. G had ongoing anxiety and anger over abandoning their homes and their unresolved life situations.

Other symptom clusters and isolated problems

These symptoms and problems occurred in fewer subjects and typically require more than a medical history to diagnose. Several are exacerbations of pre-existing conditions with obvious connections to situations of high stress or stress hormone (epinephrine, cortisol) output (cardiac arrhythmias, hypertension, irritable bowel, gastroesophageal reflux, glucose instability). One is an extension of a core symptom (unusual migraine aura). Others may indicate different kinds of direct effects of noise on body tissues, as in the vibroacoustic disease model of noise effects (respiratory infections, asthma, clotting abnormalities),[31] or other types of secondary effects (asthma).[32]

Respiratory infection/inflammation cluster. Seven subjects had unusual or prolonged lower respiratory infections during exposure (A2, B1, C2, E2, F1, F3, F4), and two of these also had prolonged asthma exacerbations (F1, F3). These two, however, were also taking a lot of paracetamol (acetaminophen) for their turbine-associated headaches. Four subjects had unusually severe or prolonged middle ear problems (C7, F2, G3, G4).

---

[31] Castelo Branco and Alves-Pereira 2004.

[32] Beasley R, Clayton T, Crane J, von Mutius E, Lai CK, Montefort S, Stewart A; ISAAC Phase Three Study Group. 2008. Association between paracetamol use in infancy and childhood, and the risk of asthma, rhinoconjunctivitis, and eczema in children aged 6–7 years: analysis from Phase Three of the ISAAC programme. Lancet 372(9643): 1039–48.

670

Cardiovascular cluster. Two subjects had exacerbations of pre-existing dysrhythmias (F1, J2). Two women had hypertension that increased during and after the exposure period, requiring medication after the end of exposure. Both still had considerable stress related to moving out and not being able to establish another regular home, and depressed husbands (C2, F2).

Gastrointestinal cluster. Four subjects had exacerbations of pre-existing gastroesophageal reflux (GER), ulcer, or irritable bowel, two with irritable bowel and upper gastrointestinal symptoms at the same time (D1, F1, F2, J2).

Arthralgia/myalgia cluster. One healthy 32-year-old woman (G2) noted pain in one elbow while in her exposed house. It resolved when she went away for vacations with her family, and recurred when she returned. It resolved quickly when the family moved away, even though she did lots of lifting during the move. A 57-year-old woman (H2) with lupus arthritis and fibromyalgia at baseline experienced painful exacerbations whenever she returned home, with return to baseline when away. A 56-year-old woman (E2) with fibromyalgia at baseline had exacerbations which resolved during times away from her exposed home and after moving.

Diabetes control. A 56-year-old man with Type II diabetes (E1), stable on oral medications and insulin before exposure, had marked glucose instability accompanied by visual blurring, retinal changes, and polyuria during exposure.

Anticoagulation. A 75-year-old woman with atrial fibrillation (F4) had stable INR values on 2–4 mcg warfarin daily for 10 years. By 16 months of exposure, her warfarin dose had been increased to 8–9 mcg daily in response to decreasing INR values.

671

Ocular cluster. Three subjects exposed to the same turbines (two men age 32–55 and one woman age 53; A1, B1, B2) had ocular pain, pressure, and/or burning synchronously with headache and tinnitus. Mr. D (D1, age 64) had a painless retinal stroke, losing half the vision in his left eye. Mr. D had a normal CT scan of the brain and was examined by an ophthalmologist.

Complex migraine phenomena. A 19-year-old fisherman (C4) with migraine at baseline had complex visual symptoms with flashes in square patterns in one eye at a time (scintillating scotoma), evolving to blurring and visual loss for 30 seconds to 2 minutes, also in one eye at a time (amaurosis fugax), right more than left, repetitively during the last month of his 15–21 month exposure until 8–12 months after exposure ended, with a decrease in frequency by 7 months after moving out. These events happened at any time of day and rarely overlapped with headaches or tinnitus. He had normal ophthalmologic exams, normal MRI and MRA scans of the brain and associated arteries, and a normal evaluation for clotting abnormalities and vasculitis. The events resolved completely with normal vision. The same man experienced repetitive complex basilar migraines with aura after the first few months of his 15–21 month turbine exposure, involving daily bilateral paresis and paresthesias of his legs and occasional headache, tinnitus, and light-headedness. The leg symptoms resolved on the same schedule as the eye symptoms, though headaches and nausea continue to be triggered regularly by seasickness.

Discussion

The core symptoms of Wind Turbine Syndrome are sleep disturbance, headache, tinnitus, other ear and hearing sensations, disturbances to balance and equilibrium, nausea, anxiety, irritability, energy loss, motivation loss, disturbances to memory and concentration, and *Visceral Vibratory Vestibular Disturbance*

672

(VVVD). Core symptoms are defined as common and widely described by study participants, closely linked in time and space to turbine exposure, and amenable to diagnosis by medical history. The latter was a particular requirement of this study. The subjects of this study had other types of health problems during exposure, discussed in "Other symptom clusters and isolated problems," but different types of study will be needed to find out if there is a link between these problems and wind turbine exposure.

The most distinctive feature of Wind Turbine Syndrome is the group of symptoms I call *Visceral Vibratory Vestibular Disturbance*. The adults who experience this describe a feeling of internal pulsation, quivering, or jitteriness, accompanied by nervousness, anxiety, fear, a compulsion to flee or check the environment for safety, nausea, chest tightness, and tachycardia. The symptoms arise day or night, interrupting daytime activities and concentration, and interrupting sleep. Wakefulness is prolonged after this type of awakening. Subjects observe that their symptoms occur in association with specific types of turbine function: the turbines turned directly towards or away from them, running particularly fast, or making certain types of noise. The symptoms create aversive reactions to bedroom and house. Subjects tend to be irritable and frustrated, especially over the loss of their ability to rest and be revitalized at home. Subjects with VVVD are also prone to queasiness and loss of appetite even when the full set of symptoms is not present.

There is no statistical association in this study between VVVD and pre-existing panic episodes (which occurred in none of the subjects) or other mental health disorders, such as depression, anxiety, bipolar disorder, or post-traumatic stress disorder. There is a highly significant association between VVVD and pre-existing motion sensitivity ($p = 0.005$).

Headaches more frequent or severe than at baseline occurred in all migraineurs in the study, and all children with headaches in the study were migraineurs or the children of migraineurs. Non-migrainous adults also got severe headaches around turbines, and indeed about half the people with headache worse than baseline (9 out of 19) were adults without history of migraine. Pre-exposure migraine is a significant risk factor for more severe or frequent headaches during turbine exposure ($p = 0.004$), but does not account for all the cases of headache.

Tinnitus occurred as a migraine aura in three subjects, but statistically in the study group tinnitus was not significantly associated with pre-existing migraine disorder, but rather with sensations of ear popping, pressure, or pain during exposure ($p = 0.008$), previous industrial noise exposure ($p = 0.013$), past history of tinnitus ($p = 0.017$), baseline permanent hearing impairment ($p = 0.040$), and (weakly) with dizziness/disequilibrium during exposure ($p = 0.058$). Like the other core symptoms, tinnitus resolved or returned to baseline when subjects were away from turbines. Previous noise exposure, past tinnitus, and baseline hearing impairment all suggest prior damage to the cochlea as a risk factor. The co-occurring symptoms of ear popping, pressure, and pain during exposure suggest that tinnitus may be caused near turbines by transient alterations in inner-ear fluid pressures (perilymph or endolymph). The weak correlation between tinnitus and dizziness/disequilibrium suggests that the proposed pressure shift may concurrently affect vestibular organ function.

Visceral Vibratory Vestibular Disturbance (VVVD)

The work of Mittelstaedt on visceral detectors of gravity,[33,34] and

[33] Mittelstaedt H. 1996. Somatic graviception. Biol Psychol 42(1–2): 53–74.

[34] Mittelstaedt H. 1999. The role of the otoliths in perception of the vertical and in path integration. Ann N Y Acad Sci 871: 334–44.

674

Balaban and others on balance-anxiety linkages,[35–39] opens a window on the VVVD symptom set. Balaban, a neuroscientist, has localized and described the neural connections among the vestibular organs of the inner ear, brain nuclei involved with balance processing, autonomic and somatic sensory inflow and outflow, the fear and anxiety associated with vertigo or a sudden feeling of postural instability, and aversive learning.[40] These form a coordinated, neurologically integrated system based in the parabrachial nucleus of the brainstem and an associated neural network.[41,42] Several aspects of this system need to be considered here.

First, there appear to be not three but four body systems for regulating balance, upright posture, and the sense of position and motion in space.[43,44] The first three systems are the eyes, the semicircular canals and otolith organs of the inner ear (vestibular organs), and somatic input from skin, skeletal muscles, tendons,

---

[35] Balaban CD, Yates BJ. 2004. The vestibuloautonomic interactions: a teleologic perspective. Chapter 7 in *The Vestibular System*, ed. Highstein SM, Fay RR, Popper AN, pp. 286–342. Springer-Verlag, New York.

[36] Balaban CD. 2002. Neural substrates linking balance control and anxiety. Physiology and Behavior 77: 469–75.

[37] Furman JM, Balaban CD, Jacob RG. 2001. Interface between vestibular dysfunction and anxiety: more than just psychogenicity. Otol Neurotol 22(3): 426–27.

[38] Balaban CD. 2004. Projections from the parabrachial nucleus to the vestibular nuclei: potential substrates for autonomic and limbic influences on vestibular responses. Brain Res 996: 126–37.

[39] Halberstadt A, Balaban CD. 2003. Organization of projections from the raphe nuclei to the vestibular nuclei in rats. Neuroscience 120(2): 573–94.

[40] Balaban and Yates 2004.

[41] Balaban CD, Thayer JF. 2001. Neurological bases for balance-anxiety links. J Anx Disord 15: 53–79.

[42] Balaban 2002.

[43] Mittelstaedt 1996.

[44] Mittelstaedt 1999.

675
EXHIBIT
23b

and joints (somatosensory system). The fourth system is visceral detection of gravity, upright position, and acceleration (meaning change in speed or direction of movement) by *visceral graviceptors*. These include stretch receptors in mesenteries or other connective tissue supporting organs or great vessels, and integrated systems of pressure detection in vessels and organs.[45] Such receptors have been localized to the kidneys and to the great vessels or their supporting structures in the mediastinum.[46] Mittelstaedt shows (by clever calculation and experimentation with people positioned in various ways on spinning centrifuge tables in the dark) that the visceral graviceptors control about 60% of our perception of position relative to gravity (meaning our sense of whether we are vertical or horizontal, or somewhere in between), compared to a 40% contribution made by the otolith organs.[47] Von Gierke (an older dean of vibration studies for the US space program) considers an inter-modality sensory conflict related to phase differences between the abdominal visceral graviceptors and the otolith organs to be a possible cause of motion sickness.[48]

The second critical element is central processing: how sensory information about motion and position is integrated by the brain, what other brain centers are activated, and what kinds of signals the brain then sends back to the body. Balaban and colleagues describe how the parabrachial nucleus network receives motion and position information from visual, vestibular (inner ear), somatosensory, and visceral sensory input, and is linked to brain

---

[45] Balaban and Yates 2004.

[46] Vaitl D, Mittelstaedt H, Baisch F. 2002. Shifts in blood volume alter the perception of posture: further evidence for somatic graviception. Int J Psychophysiol 44(1): 1–11.

[47] Mittelstaedt 1999.

[48] von Gierke HE, Parker DE. 1994. Differences in otolith and abdominal viscera graviceptor dynamics: implications for motion sickness and perceived body position. Aviat Space Environ Med 65(8): 747–51.

centers and circuits that mediate anxiety and fear, including the amygdala (a key mediator of fear reactions) and serotonin and norepinephrine-bearing neurons radiating from the midbrain.[49–51] Meaning that our sense of balance and stability in space is closely connected—neurologically—to fear and anxiety.

Balaban illustrates with a story. He asks the reader to visualize waiting in traffic on a hill for a light to turn. Out of the corner of your eye you see the truck next to you starting to inch forward, and you jam your foot on the brake, since your sensory system has told you that you are starting to slip backwards. There's a bit of panic in that moment, quickly settled as you realize you are indeed stable in space and not moving. The story illustrates how a sensation of unexpected movement elicits alerting and fear. When the sense of movement is ongoing and cannot be integrated with the evidence of the other senses, as happens in vertigo, there is a more prolonged fear reaction. In fact, as Balaban shows, the association of fear with vertigo has been known since ancient times.[52]

The third critical element is integrated neurologic outflow to the body from the parabrachial nucleus network to both the somatic (conscious, voluntary) and visceral (autonomic) effector systems. The somatic musculature is responsible for that fast foot on the brake, for righting movements of limbs, torso, and neck, and for breathing motions of the diaphragm and chest wall. The autonomic system is responsible for blood flow, heart rate, blood pressure, sweating, nausea, and other automatic, non-conscious modifications to visceral functioning. In a fear response, there is integrated outflow to these two systems—the somatic and visceral/

[49] Balaban and Thayer 2001.

[50] Balaban 2002.

[51] Halberstadt and Balaban 2003.

[52] Balaban and Thayer 2001.

autonomic. Experimental work with animals shows that vestibular signaling has profound effects on autonomic regulation of body temperature, heart rate, vascular resistance, and circadian rhythms of activity and hormone secretion.[53,54] These effects extend to humans. Vestibular stimulation by passive linear acceleration causes blood pressure and heart rate increases, with diminished responses in people with reduced vestibular function.[55]

The parabrachial nucleus network is also involved in aversive learning,[56] an experience in which nausea, if present, plays a dominant role.[57]

In VVVD, subjects detect unusual types of movement (pulsation, internal vibration, internal quivering) or other sensations (pressure, a sense of fighting something to breathe, pins and needles) in the chest or in the coordinated chest-abdominal internal space. The chest and abdomen are separated and unified by the diaphragm, which, as a striated somatic muscle, has fine-grained sensitivity to motion and stretch. The diaphragm sends signals to the brain which are specific and localizable in time and space, as opposed to visceral receptors, which send signals that are vague, like discomfort, malaise, fullness, or nausea. The diaphragm is tightly

[53] Murakami DM, Erkman L, Hermanson O, Rosenfeld MG, Fuller CA. 2002. Evidence for vestibular regulation of autonomic functions in a mouse genetic model. Proc Natl Acad Sci USA 99(26): 17078–82.

[54] Wilson TD, Cotter LA, Draper JA, Misra SP, Rice CD, Cass SP, Yates BJ. 2006. Vestibular inputs elicit patterned changes in limb blood flow in conscious cats. J Physiol 575(2): 671–84.

[55] Yates BJ, Aoki M, Burchill P, Bronstein AM. 1999. Cardiovascular responses elicited by linear acceleration in humans. Exp Brain Res 125: 476–84.

[56] Balaban and Thayer 2001.

[57] Garcia J, Ervin FR. 1968. Gustatory-visceral and telereceptor-cutaneous conditioning: adaptation in internal and external milieus. Commun Behav Biol 1: 389–415.

678

bound to one of the largest abdominal organs, the liver, and they move as a unit during breathing.

The chest, via the mouth, nose, trachea, smaller airways, and air sacs of the lungs, is open to the air. Pressure fluctuations in the air (sound waves) have free access to this airspace within the body when we breathe. Pressure fluctuations in the air also have access to the ear, which is designed to funnel them to the tympanic membrane, which concentrates their energy and transmits it to the inner ear. The ear and the chest are different size spaces with walls of different mobility and elasticity. Hence they respond differently to air pressure fluctuations (sound waves) of different sizes.

Studies of whole-body vibration focus on the easily mobile diaphragm and coupled abdominal organs. Being mobile, with the air of the lungs on one side and the soft abdominal wall on the other, this thoraco-abdominal system is easily set in motion by lower energy (amplitude) vibrations than are required to perturb other parts of the body.[58] Each part of the body has its own resonance frequency with regard to vibration. When an object is vibrated at its resonance frequency, the vibration is amplified. The resonant frequency of the thoraco-abdominal system, as it moves vertically towards and away from the lungs, lies between 4 and 8 Hz for adult humans.[59] Vibrations between 4 and 6 Hz set up resonances in the trunk with amplification up to 200%.[60] Related chest and abdominal effects are found in the same frequency range. Vibrations in the 4–8 Hz range influence breathing movements, 5–7 Hz can cause chest pains, 4–10 Hz abdominal pains, and 4–9

[58] Coermann RR, Ziegenruecker GH, Wittwer AL, von Gierke HE. 1960. The passive dynamic mechanical properties of the human thorax-abdominal system and of the whole body system. Aerosp Med 31(6): 443–55.

[59] von Gierke and Parker 1994.

[60] Hedge 2007.

679

Hz a general feeling of discomfort.[61] In small children under 40 pounds, the vertical resonance or power absorption peaks at 7.5 Hz, as opposed to 4–5 Hz for adults.[62]

Low frequency noise can cause the human body to vibrate, as quantified by researchers in Japan.[63] The degree to which the body surface is induced to vibrate by low frequency noise is correlated with subjective unpleasantness (a sensation suggesting visceral as well as surface/somatic stimulation by the noise).[64]

With this background, I propose the following mechanism for VVVD. Air pressure fluctuations in the range of 4–8 Hz, which may be harmonics of the turbine blade-passing frequency, may resonate (amplify) in the chest and be felt as vibrations or quivering of the diaphragm with its attached abdominal organ mass (liver). Slower air pressure fluctuations, which could be the blade-passing frequencies themselves or a low harmonic (1–2 Hz), would be felt as pulsations, as opposed to the faster vibrations or quivering. (The vibrations or pressure fluctuations may also be occurring at different frequencies, without this particular resonance amplification.) The pressure fluctuations in the chest could disturb visceral receptors, such as large vessel or pulmonary baroreceptors or mediastinal stretch receptors which function as visceral graviceptors. These aberrant signals from the visceral graviceptors, not concordant with signals from the other parts of the motion-detecting system, have the potential to activate

---

[61] Rasmussen 1982.

[62] Giacomin J. 2005. Absorbed power of small children. Clin Biomech 20(4): 372–80.

[63] Takahashi Y, Yonekawa Y, Kanada K, Maeda S. 1999. A pilot study on the human body vibration induced by low-frequency noise. Ind Health 37: 28–35.

[64] Takahashi Y, Kanada K, Yonekawa Y, Harada N. 2005. A study on the relationship between subjective unpleasantness and body surface vibrations induced by high-level low-frequency pure tones. Ind Health 43: 580–87, p. 580.

the integrated neural networks that link motion detection with somatic and autonomic outflow, emotional fear responses, and aversive learning. The people who are susceptible to responding in this way are those who in the past have become nauseated in response to other vertically oriented, anomalous environmental movements (seasickness or carsickness). Thus panic episodes with autonomic symptoms such as tachycardia and nausea arise during wakefulness or sleep in people with pre-existing motion sensitivity but without prior history of panic, anxiety, or other mental health disorders. Repeated triggering of these symptoms creates aversive learning, wherein the person begins to feel horror and dread of things associated with the physical sensations, such as his bedroom or house where he previously found comfort and regeneration.

VVVD was identified in the study in 14 out of 21 adult subjects. The behavior and experiences of other subjects, especially children, could be interpreted as partial manifestations of the same problem. For example, the two toddlers in the study, both age 2½ (A3, G5), had night terrors. They awoke screaming multiple times per night, and were inconsolable and difficult to get back to sleep. The little girl (G5) would fight her mother, grabbing onto the posts of the bunk bed, to avoid going back into her own bed after awakening in this state. This shows clear parallels with the fear responses, prolonged awake periods, and aversive responses of the adults with VVVD. Both toddlers were agitated and irritable in the daytime, also similar to the adults in the study. Both 5-year-olds in the study, a boy and a girl (C7, G4), also frequently woke up fearful at night.

Perturbing the inner ear

I propose that disrupted stimulation of other channels of the balance system, especially the inner-ear vestibular organs, is also likely to play a role in Wind Turbine Syndrome. Altogether, in subjects with or without VVVD, the Wind Turbine Syndrome core symptoms resemble the symptoms of a balance or vestibular disorder, meaning malfunctioning of the inner-ear motion-detecting organs (peripheral vestibular dysfunction) or of brain processing of balance-related neural signals (central balance dysfunction). These symptoms may arise near wind turbines due to abnormal stimulation of the classical balance pathways (visual, vestibular, and somatosensory), perhaps in an additive fashion if several pathways are disturbed simultaneously.

A clinical rule of thumb is that two of the three classical balance channels have to be working and producing coherent information (with agreement among channels) for a person to keep his or her balance. (How this clinical rule will incorporate the new fourth channel of balance information is yet to be seen. It may be that the sensory integrative process is actually broader, taking into account the amounts and quality of information coming from each channel, not just whether a channel is active.) The three classical pathways are 1) vision, which includes a) seeing one's orientation relative to objects and the orientation of objects relative to gravity, b) movement of images across the retina, called "retinal slip," and c) parallax or distance detection; 2) somatosensory, which involves stretch signals from muscles, tendons, and joints, and touch sensations from the skin; and 3) signals from the inner-ear vestibular organs.

The vestibular organs are 1) the semicircular canals, which detect angular acceleration during rotation of the head in any of three planes, and 2) the otolith organs (utricle and saccule), which detect gravity, tilt (static or moving), and linear accelerations by virtue of microscopic calcium carbonate crystals (otoconia) positioned in a protein matrix over the sensing hair cells. In the utricle, the patch of hair cells plus otoconia (called the macula) is oriented horizontally and is sensitive to tilts and (in upright people) to the horizontal component of linear accelerations. In the saccule, the macula is vertical, sensitive to tilts and to the vertical component of linear accelerations (including gravity) in upright people. The inner-ear or labyrinthine organs are delicate, membranous, interconnected structures with fluid inside (endolymph) and outside (perilymph), suspended in tiny canals and chambers through solid temporal bone at the base of the skull. The vertically oriented macula of the saccule is firmly bound to temporal bone over its entire area, but the horizontally oriented macula of the utricle has been recently found to be attached to temporal bone only at its anterior end,[65] a property that gives it an additional degree of freedom that may influence its tuning or resonance with regard to vibration.[66] Hair cells, which send neural signals when mechanically perturbed, are also present in specific parts of the semicircular canals and the cochlea, which is the spiral-shaped hearing organ.

In the current study, two subjects (C2, E2) were sensitive to the visual pathway with regard to triggering of symptoms. They developed severe headaches when exposed to the moving shadows of turbine blades. One (C2) had known migraine and was prone to vertigo. The other (E2) had fibromyalgia and a history of two pre-exposure episodes of vertigo. Fibromyalgia, a syndrome of chronic, diffuse pain of central origin,[67] is frequently accompanied by vertigo and dizziness.[68]

---

[65] Uzun-Coruhlu H, Curthoys IS, Jones AS. 2007. Attachment of the utricular and saccular maculae to the temporal bone. Hear Res 233(1–2): 77–85.

[66] Todd NP, Rosengren SM, Colebatch JG. 2009. A utricular origin of frequency tuning to low-frequency vibration in the human vestibular system? Neurosci Lett 451(3): 175–80.

[67] Staud R, Cannon RC, Mauderli AP, Robinson ME, Price DD, Vierck CJ Jr. 2003. Temporal summation of pain from mechanical stimulation of muscle tissue in normal controls and subjects with fibromyalgia syndrome. Pain 102: 87–95.

[68] Rosenhall U, Johansson G, Orndahl G. 1996. Otoneurologic and audiologic findings in fibromyalgia. Scand J Rehabil Med 28(4): 225–32. In this study, 72% of 168 fibromyalgia patients had dizziness or vertigo, most with abnormalities on otoneurologic testing.

683

Two subjects (C2, J2) noticed vibrations in their lower legs at certain locations on their properties, which opens the possibility of disruption of the somatosensory channel.[69] An audiologist detected vibration in the floor of an affected room in the C family's house, becoming nauseated when he put his forehead against it, an effect he interpreted as stimulation of the vestibular organs by bone conduction.[70]

I suspect that the inner-ear vestibular organs—and the cochlea—are abnormally stimulated in Wind Turbine Syndrome, especially in subjects who have marked ear symptoms such as tinnitus (including the sensation of noise inside the head) and ear pressure, popping, or pain. Families A and B, exposed to the same set of turbines, showed this pattern of symptoms especially strongly. All four adults (A1, A2, B1, B2) also had unsteadiness on their feet without accompanying vertigo or history of migraine, vertigo, prior unsteadiness, or neurologic disease. Unsteady gait, or ataxia, is generally associated with cerebellar dysfunction, but can also indicate otolith dysfunction.[71] (Vestibular nuclei in the brainstem are richly interconnected with the cerebellum.)[72] Other subjects (C2, G1, J1) had vertigo during exposure (C2 also had observed nystagmus), suggesting that disordered signals were reaching the vestibulo-ocular reflex arc from the semicircular canals or otolith organs.

---

[69] Hanes DA, McCollum G. 2006. Cognitive-vestibular interactions: a review of patient difficulties and possible mechanisms. J Vestib Res 16(3): 75–91. Vibration of calf muscles is a method sometimes used in balance studies to simulate somatosensory disturbance, p. 77.

[70] Noise report prepared for family C, May 2006.

[71] Schlindwein P, Mueller M, Bauermann T, Brandt T, Stoeter P, Dieterich M. 2008. Cortical representation of saccular vestibular stimulation: VEMPs in fMRI. Neuroimage 39: 19–31.

[72] Colebatch JG, Halmagyi GM, Skuse NF. 1994. Myogenic potentials generated by a click-evoked vestibulocollic reflex. J Neurol Neurosurg Psychiatry 57(2): 190–97.

In Wind Turbine Syndrome, I hypothesize that low frequency noise or vibration impinges on the delicately mobile labyrinthine organs, but not in a way that stimulates the cochlea to a coherent representation of sound. Instead, the low frequency noise or vibration, I suggest, may stimulate various parts of the labyrinth in a disorganized fashion, experienced as tinnitus from the cochlea, a distorted sense of vertical from the otolith organs, or illusory self-motion from the otolith organs or semicircular canals. The dominant sensory impression may depend on 1) the frequencies and intensities of low frequency noise and vibration coming from the turbines, 2) whether the noise or vibration arrives at the ear through the air and outer/middle ear or is bone-conducted, and 3) the susceptibilities and prior histories of the subjects, such as migraine with its tendency towards vertigo, prior damage to the cochlea, or other conditions or anomalies of the inner ear.[73]

The statistical correlation in the current study between tinnitus and ear popping, pressure, or pain during exposure suggests a refinement to this mechanism: altered fluid pressure relationships in the inner ear may distort cochlear mechanics during exposure and cause tinnitus, and distort utricular and saccular mechanics to create instability or ataxia and other second-order vestibular symptoms.

Low frequency noise, in fact, is known to distort endolymphatic pressure and volume after just short exposures to loud but not

[73] For example, dehiscence of the superior semicircular canal, in which alterations in inner-ear pressure relationships due to a "third window" effect (from an abnormal hole in the bone between the superior semicircular canal and the cranial cavity) cause conductive hearing loss, increased sensitivity to bone-conducted sound or vibration, and the tendency to become unbalanced by sounds (Tullio effect). Dislocation of the stapes footplate, labyrinthine fistulas, and endolymphatic hydrops can also underlie the Tullio phenomenon. (See Colebatch JG, Day BL, Bronstein AM, Davies RA, Gresty MA, Luxon LM, Rothwell JC. 1998. Vestibular hypersensitivity to clicks is characteristic of the Tullio phenomenon. J Neurol Neurosurg Psychiatry 65: 670–78.)

damaging low frequency tones.[74] This temporary effect is associated with hyperacusis, a distortion of hearing function in which sounds are perceived as louder.[75] One subject in the current study, G2, had hyperacusis while living near turbines, and another (C2) noticed hyperacusis after her tinnitus resolved, after she moved away from the turbines. Tinnitus may also be associated with increased perilymphatic and intracranial pressure in the presence of an open cochlear aqueduct, which provides a direct channel linking these two fluid spaces.[76]

There is both animal and human precedent for thinking that certain types of environmental noise or vibration may stimulate the otolith organs and cause disturbance to motion and position sense. Vestibular organ structures have been conserved during evolution, meaning they are rather similar in fish, amphibians, and other vertebrate taxa, including humans. All the vertebrates have semicircular canals and otolith organs. Like us, fish use their otolith organs (utricle, saccule, and an extra one, the lagena) to sense linear accelerations and tilt relative to gravity, but these organs in "non-specialist" fish species (such as cod) are also the fishes' auditory organs. The otolith organs in these fish are highly sensitive to nearby perturbations in the water ("near-field sound")[77] with peak sensitivities in the low frequency range between 40 and 120 Hz.[78] Atlantic cod otolith organs are so sensitive to

[74] Salt AN. 2004. Acute endolymphatic hydrops generated by exposure of the ear to nontraumatic low-frequency tones. J Assoc Res Otolaryngol 5(2): 203–14.

[75] Salt 2004.

[76] Reid A, Cottingham CA, Marchbanks RJ. 1993. The prevalence of perilymphatic hypertension in subjects with tinnitus: a pilot study. Scand Audiol 22: 61–63.

[77] Sand O, Karlsen HE, Knudsen FR. 2008. Comment on "Silent research vessels are not quiet" [J Acoust Soc Am 2007; 121(4): EL145–50]. J Acoust Soc Am 123(4): 1831–33.

[78] Fay RR, Simmons AM. 1999. The sense of hearing in fishes and amphibians. In *Comparative Hearing: Fish and Amphibians*, ed. Fay RR, Popper AN, pp. 269–317. Springer-Verlag, New York.

infrasound in water (at 0.1 Hz, or one wave every 10 seconds) that the fish may be able to use seismic sounds from the Mid-Atlantic Ridge or the sounds of waves breaking on distant shores, or even more complex mechanisms, to guide them during migration.[79,80] Directional infrasound detection plays a role in predator avoidance behaviors.[81]

In humans, there is a substantial body of experimental evidence showing that both air-conducted sound and bone-conducted sound (vibration) stimulate the otolith organs and cause measurable impacts on vestibular reflexes, independent of their stimulation of the cochlea. Air-borne sound in the form of loud clicks or short tone bursts induces inhibitory neural signals in the sternocleidomastoid muscles in the anterior neck. Called the *vestibular evoked myogenic potential* (VEMP), this is an extremely fast or "short-latency" neural response that is part of the vestibulo-collic reflex.[82] Bone-conducted sound or vibration is more efficient than air-conducted clicks or tones at stimulating the otolith organs: both the absolute decibel levels and decibels above hearing threshold needed to produce the VEMP response are lower for bone-conducted sound.[83]

Studies of both the VEMP and—a second measure of vestibular function—the *ocular vestibular evoked myogenic potential* (OVEMP) show that the tuning (best frequency response) for both

---

[79] Sand O, Karlsen HE. 1986. Detection of infrasound by the Atlantic cod. J Exp Biol 125: 197–204.

[80] Sand O, Karlsen HE. 2000. Detection of infrasound and linear acceleration in fishes. Phil Trans R Soc Lond B 355: 1295–98.

[81] Karlsen HE, Piddington RW, Enger PS, Sand O. 2004. Infrasound initiates directional fast-start escape responses in juvenile roach Rutilus rutilus. J Exp Biol 207(Pt 24): 4185–93.

[82] Colebatch et al. 1994.

[83] Welgampola MS, Rosengren SM, Halmagyi GM, Colebatch JG. 2003. Vestibular activation by bone conducted sound. J Neurol Neurosurg Psychiatry 74: 711–18.

VEMP and OVEMP for air-conducted sound lies between 400 and 800 Hz.[84] Whereas with bone-conducted sound (vibration), the best frequency response for both VEMP and OVEMP is at 100 Hz. Modeling of the frequency tuning and other aspects of the response, such as laterality, phase differences, and gain, suggests that the air-conducted peak comes from the rigidly attached saccule, whereas the bone-conducted or vibratory peak derives from the more mobile utricle.[85] A particular type of vestibular hair cell, Type I cells, is thought to be involved in the utricular response and accounts for the marked sensitivity of the OVEMP response to vibration, since these cells typically produce a strong neural vestibular signal in response to a low degree of mechanical disturbance.[86,87]

Most exciting, Todd et al. provide direct experimental evidence that at the 100 Hz tuning peak, the vestibular organs (probably utricle, as above) of normal humans are ***much more sensitive than the cochlea*** to low frequency bone-conducted sound/vibration.[88] The researchers applied vibration directly to the skin over the bony mastoid prominence behind the subjects' ears, adjusting the power by measuring the tiny whole-head acceleration produced by each vibration force and frequency. They were able to elicit and measure neural signals of the vestibulo-ocular reflex (OVEMP, as above) at vibration intensities 15 dB below the subjects' hearing thresholds. In other words, the amount of vibration/bone-conducted sound was so small that the subjects could not hear it, yet the vestibular parts of their inner ears still responded to the vibration and

[84] Todd et al. 2009.

[85] Todd et al. 2009.

[86] Todd et al. 2009.

[87] Curthoys IS, Kim J, McPhedran SK, Camp AJ. 2006. Bone conducted vibration selectively activates irregular primary otolithic vestibular neurons in the guinea pig. Exp Brain Res 175(2): 256–67.

[88] Todd et al. 2008.

transmitted signals into the balance and motion networks in the brain, resulting in specific types of eye muscle activation. Since dB is a base 10 logarithmic measure, *15 dB below* means a signal 0.0316 ($10^{-1.5}$), or about 3%, of the power or amplitude of the signal these normal subjects could hear.

The researchers note that "the very low thresholds we found are remarkable as they suggest that humans possess a frog- or fish-like sensory mechanism which appears to exceed the cochlea for detection of substrate-borne low-frequency vibration and which until now has not been properly recognized."[89] Thus the potential exists, in normal humans, for stimulation of balance signals from the inner ear by low frequency noise and vibration, even when the noise or vibration does not seem especially loud, or even cannot be heard. In the presence of pre-existing inner-ear pathology, thresholds for vestibular stimulation by noise or vibration are even lower than in normal subjects.[90]

Central balance processing

When there is conflict in neurologically normal people among the signals coming from the different balance channels, the brain areas that integrate the information quickly compensate by suppressing or down-weighting information from the anomalous channel[91]—information that does not match what is coming from the other channels. On functional brain scans, vestibular and visual cortical areas show a pattern of inverse activation and deactivation, such

[89] Todd et al. 2008, p. 41.

[90] Colebatch et al. 1998. See footnote 73.

[91] Jacob RG, Redfern MS, Furman JM. 2009. Space and motion discomfort and abnormal balance control in patients with anxiety disorders. J Neurol Neurosurg Psychiatry 80(1): 74–78. E-pub 2008 July 24.

689

that vestibular activation deactivates visual cortex and vice versa.[92,93] In people with vestibular organ damage, long-term compensation promotes reliance on vision ("visual dependence") or on somatosensory input from muscles, tendons, joints, and skin ("surface dependence"). A visually dependent vestibular patient cannot adequately suppress visual input and up-weight vestibular signals because of pre-existing problems with the vestibular channel,[94] leaving the person dependent on visual perception of motion and position even in environments where the visual information is ambiguous. When combined with the sense of fear generated by a feeling of postural instability or uncertainty (as reviewed above), this can create fear of heights.

It can also cause Space and Motion Discomfort,[95] a condition in which situations challenging to motion and position sense create discomfort. These situations include looking up at tall buildings, scanning shelves in a supermarket, closing eyes in the shower, leaning far back in a chair, driving through tunnels, riding in an elevator, riding in the back seat of a car, or reading in the car.[96]

Even without vestibular organ disease, some people have Space and Motion Discomfort due to a central or brain-based difficulty with

---

[92] Brandt T, Bartenstein P, Janek A, Dieterich M. 1998. Reciprocal inhibitory visual-vestibular interaction. Visual motion stimulation deactivates the parieto-insular vestibular cortex. Brain 121(Pt. 9): 1749–58.

[93] Brandt T, Dieterich M. 1999. The vestibular cortex: its locations, functions, and disorders. Ann NY Acad Sci 871: 293–312.

[94] Redfern MS, Yardley L, Bronstein AM. 2001. Visual influences on balance. J Anxiety Disord 15(1–2): 81–94.

[95] Jacob RG, Woody SR, Clark DB, Lilienfeld SO, Hirsch BE, Kucera GD, Furman JM, Durrant JD. 1993. Discomfort with space and motion: a possible marker of vestibular dysfunction assessed by the Situational Characteristics Questionnaire. J Psychopathol Behav Assess 15(4): 299–324.

[96] Jacob et al. 2009. As a rural physician, I might also ask patients about driving past rows of parallel trees, especially with the low winter sun flashing between the trunks, as the rural equivalent of looking at lights on the wall of a tunnel.

690

the process of integrating balance signals into a coherent, moment-to-moment representation of their motion and orientation in space. Balance testing using posturography shows that such people have difficulty down-weighting anomalous information from either the visual or somatosensory channel, or have a mild, central disorder of balance control with increased postural sway even under non-challenging conditions.[97–99]

Space and Motion Discomfort is common in patients with anxiety disorders,[100,101] migrainous vertigo,[102] and migraine-anxiety related dizziness.[103] Vertigo is especially characteristic of migraine and may at times occur as a migraine aura with or without headache.[104] In one study, dizziness or vertigo was found in 54% of 200 migraine patients, half of whom also had a history of motion sickness, compared with 30% of people with tension-type headaches.[105] In a study of 72 patients with isolated recurrent vertigo, 61% were found to have migraine, compared to 10% in a control group of orthopedic patients.[106] Abnormal balance testing

---

[97] Redfern MS, Furman JM, Jacob RG. 2007. Visually induced postural sway in anxiety disorders. J Anxiety Disord 21(5): 704–16. NIH Public Access Author Manuscript, pp. 1–14.

[98] Jacob et al. 2009.

[99] Furman JM, Balaban CD, Jacob RG, Marcus DA. 2005. Migraine-anxiety related dizziness (MARD): a new disorder? J Neurol Neurosurg Psychiatry 76: 1–8.

[100] Jacob et al. 2009.

[101] Redfern et al. 2007.

[102] Neuhauser H, Leopold M, von Brevern M, Arnold G, Lempert T. 2001. The interactions of migraine, vertigo, and migrainous vertigo. Neurology 56: 436–41.

[103] Furman et al. 2005.

[104] Furman et al. 2005.

[105] Kayan A, Hood JD. 1984. Neuro-otological manifestations of migraine. Brain 107: 1123–42.

[106] Lee H, Sohn SI, Jung DK, Cho YW, Lim JG, Yi SD, Yi HA. 2002. Migraine and isolated recurrent vertigo of unknown cause. Neurol Res 24(7): 663–65.

691

is seen in patients with migraine but not in those with tension-type headaches.[107] Balance testing shows that both central and vestibular organ balance problems are found in migraine patients, especially in those who experience dizziness or vertigo.[108]

About 50% of migraine sufferers in general have histories of motion sickness, compared to only about 20% in people with tension headaches.[109] Motion sickness is the most common vestibular symptom in migraine. Motion sickness is provoked by excessively moving environments (amusement park rides, boats in rough water, airplanes in turbulence, the back of a school bus) or situations of conflict among visual, vestibular, somatosensory, and visceral signals to the balance system (reading in the car, riding in the back seat, driving in snow, simulators, IMax movies, computer images and games, space travel). The nausea of motion sickness may be accompanied by dizziness, cold sweat, pallor, headache, increased salivation, sleepiness, and apathy or disinclination for physical or mental work, thus sharing many symptoms with migraine.[110] Like migraine, motion sickness is more common in women.[111] Visual migraine aura without headache is increased in adults with a history of childhood motion sickness. Motion sickness is not associated with peripheral vestibular disorders, however, such as benign paroxysmal positional vertigo, Meniere's disease, or vestibular neuritis.[112]

---

[107] Ishizaki K, Mori N, Takeshima T, Fukuhara Y, Ijiri T, Kusumi M, Yasui K, Kowa H, Nakashima K. 2002. Static stabilometry in patients with migraine and tension-type headache during a headache-free period. Psychiatry Clin Neurosci 56(1): 85–90.

[108] Furman et al. 2005.

[109] Marcus DA, Furman JM, Balaban CD. 2005. Motion sickness in migraine sufferers. Expert Opin Pharmacother 6(15): 2691–97.

[110] Marcus et al. 2005.

[111] Marcus et al. 2005.

[112] Marcus et al. 2005.

692

The dizziness associated with anxiety disorders is not necessarily caused by the anxiety, as is often assumed in clinical practice, but may have a component of disturbed balance control.[113,114] For example, the presence of panic or fear of heights is significantly associated with abnormalities on caloric testing, a form of vestibular testing.[115] A positive result on a questionnaire for Space and Motion Discomfort is significantly associated with abnormality on posturography showing either surface[116] or visual[117] dependence. In testing of vestibulo-ocular reflexes, anxiety patients have been found to have higher vestibular sensitivity or gain than normal controls.[118] Balance assessments of patients diagnosed with panic attacks or agoraphobia (fear of leaving the house) show a high proportion with abnormalities of vestibular function, in some studies greater than 80%, especially if the patients have episodes of dizziness between panic attacks.[119–122]

---

[113] Furman et al. 2005.

[114] Eckhardt-Henn A, Breuer P, Thomalske C, Hoffmann SO, Hopf HC. 2003. Anxiety disorders and other psychiatric subgroups in patients complaining of dizziness. J Anxiety Disord 17(4): 369–88.

[115] Jacob et al. 2009.

[116] Jacob et al. 2009.

[117] Redfern et al. 2007.

[118] Furman JM, Redfern MS, Jacob RG. 2006. Vestibulo-ocular function in anxiety disorders. J Vestib Res 16: 209–15.

[119] Perna G, Dario A, Caldirola D, Stefania B, Cesarani A, Bellodi L. 2001. Panic disorder: the role of the balance system. J Psychiatr Res 35(5): 279–86.

[120] Jacob RG, Furman JM, Durrant JD, Turner SM. 1996. Panic, agoraphobia, and vestibular dysfunction. Am J Psychiatry 153(4): 503–12.

[121] Yardley L, Britton J, Lear S, Bird J, Luxon LM. 1995. Relationship between balance system function and agoraphobic avoidance. Behav Res Ther 33(4): 435–39.

[122] Yardley L, Luxon LM, Lear S, Britton J, Bird J. 1994. Vestibular and posturographic test results in people with symptoms of panic and agoraphobia. J Audiol Med 3: 58–65.

Thus problems with balance function can be due to abnormalities of the inner-ear vestibular organs (utricle, saccule, and semicircular canals) or to abnormal central (brain) integration of balance signals. Mild (mostly central) abnormalities are common and associated with common conditions such as migraine, motion sensitivity, vertigo, and several types of anxiety disorder. People with mild balance abnormalities only feel off balance or insecure in challenging situations where the available sensory information is inadequate or confusing, such as at heights or in the situations described in the questionnaire for Space and Motion Discomfort. The rest of the time, people with mild, compensated balance deficits feel normal and securely oriented in space.

However, if a person is already in a state of adaptation to an ongoing vestibular organ or central balance deficit—even mild, fully compensated deficits—he or she is at particular risk for decompensation with exposure to new balance challenges. Many of the affected people in the present study, I suspect, were in this condition, because their medical histories reveal a variety of risks for mild baseline balance dysfunction. These risks include motion sensitivity, migraine disorder, prior damage to inner-ear organs from industrial noise exposure or chemotherapy, autoimmune disease,[123] fibromyalgia,[124] and normal aging (over 50). We may also consider normal early childhood (age 1–4 or so) as a time of natural mild balance dysfunction[125,126] (see discussion at the end

[123] Rinne T, Bronstein AM, Rudge P, Gresty MA, Luxon LM. 1998. Bilateral loss of vestibular function: clinical findings in 53 patients. J Neurol 245(6–7): 314–21.

[124] Rosenhall U, Johansson G, Orndahl G. 1996. Otoneurologic and audiologic findings in fibromyalgia. Scand J Rehabil Med 28(4): 225–32.

[125] Foudriat BA, Di Fabio RP, Anderson JH. 1993. Sensory organization of balance responses in children 3–6 years of age: a normative study with diagnostic implications. Int J Pediatr Otorhinolaryngol 27(3): 255–71.

[126] Steindl R, Kunz K, Schrott-Fischer A, Scholtz AW. 2006. Effect of age and sex on maturation of sensory systems and balance control. Dev Med Child Neurol 48(6): 477–82.

of the next section). Other potential risks for chronic balance deficits, not seen in this study, are whiplash injury and head injury, including concussions and milder head impacts without loss of consciousness,[127–129] and chronic inner-ear conditions such Meniere's disease, dehiscence of the superior semicircular canal, and others.[130]

Cognition and vestibular function

It is now becoming apparent that a variety of cognitive functions depend on coherent vestibular signaling. Clinicians who work with balance-disordered patients are familiar with their struggles with short-term memory, concentration, multitasking, arithmetic, and reading.[131,132] In the perilymphatic fistula syndrome, for example (a form of inner-ear pathology that can follow whiplash, minor head injuries, or pressure trauma to the ear), symptoms of dizziness, headache, stiff neck, and disturbed sleep are accompanied by marked mental performance deficits compared to the patient's baseline.[133] Such cognitive symptoms are difficult to evaluate clinically and are often dismissed as psychological in origin.[134] However, recent research using imaging and other modalities shows that vestibular function exerts a powerful influence over human thinking and memory.

---

[127] Grimm RJ, Hemenway WG, Lebray PR, Black FO. 1989. The perilymph fistula syndrome defined in mild head trauma. Acta Otolaryngol Suppl 464: 1–40.

[128] Ernst A, Basta D, Seidl RO, Todt I, Scherer H, Clarke A. 2005. Management of posttraumatic vertigo. Otolaryngol Head Neck Surg 132(4): 554–58.

[129] Claussen CF, Claussen E. 1995. Neurootological contributions to the diagnostic follow-up after whiplash injuries. Acta Otolaryngol Suppl 520, Pt. 1: 53–56.

[130] Colebatch et al. 1998.

[131] Hanes and McCollum 2006.

[132] Grimm et al. 1989.

[133] Grimm et al. 1989.

[134] Hanes and McCollum 2006.

695

The vestibular system is ancient in the vertebrate lineage. Hence its neural connections ramify widely in both older and more recently evolved parts of the brain, including the brainstem, midbrain, cerebellum, and occipital, parietal, and frontal cortex.[135] Vestibular injury causes specific cognitive difficulties, but not general cognitive impairment.[136] Vestibular effects on cognition are often attributed to competing stimuli (meaning, challenges to movement and position sense draw attention away from cognitive tasks), but may actually reflect the direct dependence of certain cognitive operations on the vestibular system.[137]

Vestibular input is critical for spatial thinking, body and spatial awareness, spatial memory, and complex spatial or map calculations.[138] Dynamic, active vestibular signaling is needed during the acquisition, storage, and use of information with spatial components, such as building mental maps or deducing a novel path between two points.[139] Patients with 5–10 year histories of bilateral vestibular loss showed marked deficits in a classic experimental task of spatial memory and navigation, accompanied, on average, by a 16.9% volume loss in the hippocampus (a temporal lobe structure essential for learning and memory).[140] In a test of general memory, however, these patients were no different from controls.[141] Vestibular signaling to the hippocampus is known to occur in both humans and other primates via a direct, two-neuron

[135] Dieterich M, Brandt T. 2008. Functional brain imaging of peripheral and central vestibular disorders. Brain 131(10): 2538–52.

[136] Hanes and McCollum 2006.

[137] Hanes and McCollum 2006.

[138] Hanes and McCollum 2006.

[139] Brandt T, Schautzer F, Hamilton DA, Bruning R, Markowitsch HJ, Kalla R, Darlington C, Smith P, Strupp M. 2005. Vestibular loss causes hippocampal atrophy and impaired spatial memory in humans. Brain 128: 2732–41.

[140] Brandt et al. 2005.

[141] Brandt et al. 2005.

696

linkage through the posterior thalamus. There are also other proposed neural pathways.[142]

Disordered vestibular input increases error rates in purely mental tasks based on visualization of remembered objects, showing that coherent vestibular input is critical for thinking successfully and efficiently in spatial terms.[143] This is true even without using sight and beyond the period of memory storage. The tasks included detailed visualization, considered an occipital (visual) cortical task, and mental rotation, a parietal cortical task.[144]

Vestibular stimulation in both humans and other primates activates a variety of areas in the parietal cortex, including 1) a core vestibular processing area (posterior insula), 2) the somatosensory strip, 3) areas involved in hemineglect in stroke patients (ventral parietal), and 4) a region "known to be involved in multimodal coordinate transformations and representation of space" (intraparietal sulcus), which is a principal site for arithmetic and counting tasks.[145]

Hemineglect is a condition after right-sided parietal stroke in which a patient can have so much unawareness of the left side of space that he is oblivious to his own left-sided body parts being paralyzed, for example, or undressed. Vestibular stimulation temporarily corrects or improves this unawareness, in ways that suggest stimulation not only to general attention, but also to cerebral structures involved

---

142 Brandt et al. 2005.

143 Mast FW, Merfeld DM, Kosslyn SM. 2006. Visual mental imagery during caloric vestibular stimulation. Neuropsychologia 44(1): 101–9.

144 Mast et al. 2006. I wonder whether the detailed visualization task also included a parietal component, given the quantitative comparison the subjects had to make with the remembered image.

145 Hanes and McCollum 2006, p. 82.

in the mental representation of space.[146,147] Vestibular stimulation also improves hemineglect patients' performance on tasks of visual localization and visual-spatial memory retrieval. At baseline, and again 24 hours after the experiment, their responses were biased away from the left side, but this bias was corrected or improved immediately after left vestibular stimulation.[148]

Studies of hemineglect patients have further shown that many mental operations are "spatialized" and dependent on parietal brain areas that have been lost, including mathematical operations involving a "mental number line" with lower numbers on the left,[149,150] clock representations of time,[151] and spelling at the beginnings (left) or ends (right) of words (errors occur opposite to the side of the parietal lesion).[152] In right-handed patients with right parietal strokes, there is no impairment to simple numeric calculation (a left-sided parietal function), but there is impairment to spatialized mathematical thinking, such as finding the midpoint between two numbers.[153] At the other extreme of mental functioning, it has been found that great mathematicians think of numbers in spatial terms,[154] which "may be more efficient because

---

[146] Geminiani G, Bottini G. 1992. Mental representation and temporary recovery from unilateral neglect after vestibular stimulation. J Neurol Neurosurg Psychiatry 55(4): 332–33.

[147] Cappa S, Sterzi R, Vallar G, Bisiach E. 1987. Remission of hemineglect and anosognosia during vestibular stimulation. Neuropsychologia 25: 775–82.

[148] Geminiani and Bottini 1992.

[149] Zorzi M, Priftis K, Umilta C. 2002. Brain damage: neglect disrupts the mental number line. Nature 417: 138–39.

[150] Vuilleumier P, Ortigue S, Brugger P. 2004. The number space and neglect. Cortex 40(2): 399–410.

[151] Vuilleumier et al. 2004.

[152] Hillis HE, Caramazza A. 1995. Spatially specific deficits in processing graphemic representations in reading and writing. Brain Lang 48 (3): 263–308.

[153] Zorzi et al. 2002.

[154] Hadamard J. 1996. *The Mathematician's Mind: The Psychology of Invention in the Mathematical Field.* Princeton University Press, NJ. In Zorzi et al. 2002.

it is grounded in the actual neural representation of numbers."[155] A recent study of outstanding human memorizers shows that spatially oriented strategies are also critical to good memory, by providing an efficient framework for memory organization and retrieval.[156]

Thus current research shows that coherent vestibular neural input is critical for spatialized forms of thinking and memory. Spatialized thinking and memory is intrinsic to many of the things we do with our minds, including mathematical thinking and memory organization (as discussed above) and many forms of map-based or visually based problem-solving or short-term memory we do in everyday life. Spatial thinking is used, for example, to figure out the most efficient path for a set of errands, remember the path and images of the items to be obtained, search for the items on the shelf, and judge if one was given the correct change. It is used for mental "maps" or calendars of one's day, week, or month and its appointments, to picture in three dimensions how to put something together, or imagine what has gone wrong inside a device and initiate a repair. It is used, as well, for understanding the visual clues and images in a movie or TV show. In this context, it is easy to see how vestibular disturbance might impair concentration (which means the ability to perform thinking tasks successfully and efficiently) and memory. Vestibular disturbance also has the potential to affect reading directly, via the reflex control exerted by semicircular canal and otolith organs over eye movements (vestibulo-ocular reflex).

Effects on concentration and memory were nearly ubiquitous in the present study, if one includes all subjects that told me about any problems in this area. For some subjects the deficits were

---

155 Zorzi et al. 2002.

156 Maguire EA, Valentine ER, Wilding JM, Kapur N. 2003. Routes to remembering: the brains behind superior memory. Nat Neurosci 6(1): 90–95.

dramatic compared to pre-exposure baseline, including the 7 out of 10 school-age children and teens who showed a decline in their academic performance. Detrimental effects on concentration and memory were significantly associated with normal memory at baseline ($p = 0.027$) and with fatigue and loss of energy and motivation during exposure ($p = 0.018$). Though sleep deprivation/disturbance undoubtedly plays a role in the problems with concentration and memory, qualitative aspects of the mental performance deficiencies suggest a mechanism other than sleep disturbance alone. I propose that this mechanism is the effect of vestibular disturbance on cognition.

It is interesting here to examine a possible role of vestibular disturbance in the learning of very young children, in the toddler and preschool years. Mrs. G (G2) volunteered that her 2½-year-old's (G5) irritability during turbine exposure was especially triggered by her older siblings' "unsteadying her" or coming so close that she thought she might be unsteadied. Children at this age are learning to keep their balance through a variety of different kinds of activities and postures. They are both fascinated and relaxed by vestibular stimulation (swinging, spinning, rolling, somersaults, etc.) and they actively explore the physical world through this play. The behavior of objects in gravity is another source of fascination, starting with babies' casting behavior and moving on to pouring water, sliding down slides, rolling things down inclines, building dams, floating toy boats, blowing bubbles, releasing helium balloons, etc. Vestibular input and processing play a critical role in a) balance during movement, b) the generation, storage, and use of internal maps, and c) recognition of the behavior of objects under the influence of gravity. Indovina et al. measured brain activity by functional MRI in adults as they watched the movement of simulated objects, finding that the vestibular network was selectively engaged when the acceleration of an object was consistent with natural

700

dramatic compared to pre-exposure baseline, including the 7 out of 10 school-age children and teens who showed a decline in their academic performance. Detrimental effects on concentration and memory were significantly associated with normal memory at baseline ($p = 0.027$) and with fatigue and loss of energy and motivation during exposure ($p = 0.018$). Though sleep deprivation/ disturbance undoubtedly plays a role in the problems with concentration and memory, qualitative aspects of the mental performance deficiencies suggest a mechanism other than sleep disturbance alone. I propose that this mechanism is the effect of vestibular disturbance on cognition.

It is interesting here to examine a possible role of vestibular disturbance in the learning of very young children, in the toddler and preschool years. Mrs. G (G2) volunteered that her 2½-year-old's (G5) irritability during turbine exposure was especially triggered by her older siblings' "unsteadying her" or coming so close that she thought she might be unsteadied. Children at this age are learning to keep their balance through a variety of different kinds of activities and postures. They are both fascinated and relaxed by vestibular stimulation (swinging, spinning, rolling, somersaults, etc.) and they actively explore the physical world through this play. The behavior of objects in gravity is another source of fascination, starting with babies' casting behavior and moving on to pouring water, sliding down slides, rolling things down inclines, building dams, floating toy boats, blowing bubbles, releasing helium balloons, etc. Vestibular input and processing play a critical role in a) balance during movement, b) the generation, storage, and use of internal maps, and c) recognition of the behavior of objects under the influence of gravity. Indovina et al. measured brain activity by functional MRI in adults as they watched the movement of simulated objects, finding that the vestibular network was selectively engaged when the acceleration of an object was consistent with natural gravity, even though the stimulus was only visual.[157] The authors use this as evidence that "predictive mechanisms of physical laws of motion are represented in the human brain"[158] under the influence of vestibular signaling of the vector of gravity. I suggest that these representations of the physical laws of motion are embedded in the human brain during early childhood as toddlers and children learn through experimentation (play) about the behavior of their bodies and other objects in gravity, and that coherent vestibular signaling is critical to this learning.

Environmental noise, learning, sleep, and health effects

Many studies have quantified the effects of environmental noise on children's learning. Reading acquisition—a language-intensive process—is especially sensitive to the effects of noise in school and at home. The effect is distinct from the effects of noise on attention or working memory,[159] and is correlated with measures of language processing such as speech recognition.[160] Airplane noise, which has a large low frequency component, has a stronger effect than traffic noise in some studies,[161] but traffic noise is also shown to have modest effects on memory in quieter communities.[162] Most studies are cross-sectional, but a longitudinal or cohort study, done

---

[157] Indovina I, Maffei V, Bosco G, Zago M, Macaluso E, Lacquaniti F. 2005. Representation of visual gravitational motion in the human vestibular cortex. Science 308: 416–19.

[158] Indovina et al. 2005.

[159] Haines MM, Stansfeld SA, Job RFS, Berglund B, Head J. 2001. A follow-up study of effects of chronic aircraft noise exposure on child stress responses and cognition. Int J Epidemiol 30: 839–45.

[160] Evans GW, Maxwell L. 1997. Chronic noise exposure and reading deficits: the mediating effects of language acquisition. Environ Behav 29(5): 638–56.

[161] *Clark C, Martin R, van Kempen E, Alfred T, Head J, Davies HW, Haines MM,* Barrio IL, Matheson M, Stansfeld SA. 2005. Exposure-effect relations between aircraft and road traffic noise exposure at school and reading comprehension: the RANCH project. Am J Epidemiol 163: 27–37.

[162] Lercher P, Evans GW, Meis M. 2003. Ambient noise and cognitive processes among primary schoolchildren. Environ Behav 35(6): 725–35.

702

when an airport was closed in one location and opened in another, showed similar effects on reading acquisition.[163] One study showed effects of noise on reading and auditory processing in children who lived in an apartment building next to a busy highway. The higher they lived in the building, the quieter were their apartments and the better their reading and auditory discrimination scores (e.g., distinguishing *goat* from *boat*). After controlling for parental education and income, the auditory discrimination scores largely explained the noise-reading linkage.[164] These effects on reading occur at sound levels far less than those needed to produce hearing damage.[165] Children with pre-existing reading deficiencies and children at higher grade levels are more affected, and longer exposure produces larger deficits.[166]

Effects suggestive of wind turbine noise impact on auditory discrimination or central auditory processing were found in the current study. During the period immediately after moving away from turbines and the cessation of her tinnitus, Mrs. A (A2, age 33) found she had a new difficulty understanding conversation in crowded, noisy places. Her son (A3, age 2½) began to confuse several consonant sounds during exposure, and continued to do so in the immediate post-exposure period.

Studies of adults in industrial settings have shown effects of noise on cognitive function when the noise is not considered loud and is nowhere near the threshold for causing damage to hearing. Polish

---

[163] Hygge S, Evans GW, Bullinger M. 2002. A prospective study of some effects of aircraft noise on cognitive performance in schoolchildren. Psychol Sci 13: 469–74.

[164] Cohen S, Glass DC, Singer JE. 1973. Apartment noise, auditory discrimination, and reading ability in children. J Exp Soc Psychol 9: 407–22.

[165] Evans GW. 2006. Child development and the physical environment. Annu Rev Psychol 57: 423–51.

[166] Evans 2006, p. 426.

703

researchers exposed workers to 50 dBA broadband noise or 50 dBA broadband noise with low frequency components (10-250 Hz) as they worked on standard psychological tests. Low frequency noise impaired performance more than broadband noise without low frequency components, especially in subjects who rated themselves as highly sensitive to low frequency noise. There was no difference in the annoyance ratings for the two types of noise, nor evidence of either habituation or sensitization.[167]

Sleep deprivation is a primary focus of studies of community noise in general and was a major factor for the subjects in the current study. The occurrence of VVVD contributes a distinctive quality to sleep disturbance and to the extent of sleep deprivation near wind turbines, since waking up in a physiologic state of panic leads to prolonged wakefulness or not returning to sleep at all. A second distinctive quality of wind turbine-associated sleep disturbance was nocturia (getting up repeatedly at night to urinate), mostly in adult women, and nocturnal enuresis (bed-wetting) in a 5-year-old girl. Nocturia resolved immediately when subjects slept away from turbines. For the 5-year-old, the enuresis stopped during a family vacation, resumed on return home, and resolved fully when the family moved away.

Studies of whole-body vibration identify 10–18 Hz as frequencies likely to create the urge to urinate,[168] a possible mechanism for nocturia during exposure. Nocturnal enuresis may be a manifestation of the same direct vibratory stimulation in a child not yet developmentally ready to awaken to bladder signals, or it may instead be a parasomnia (like sleep walking, sleep talking, and night terrors) that occurs during disordered partial arousal

[167] Pawlaczyk-Luszczynska M, Dudarewicz A, Waszkowska M, Szymczak W, Sliwinska-Kowalska M. 2005. The impact of low-frequency noise on human mental performance. Int J Occup Med Environ Health 18(2): 185–98.

[168] Rasmussen 1982.

204

from the deeper stages of sleep. Perilymphatic fistula syndrome, a vestibular disorder, includes nocturnal enuresis in adult women in its list of parasomnic manifestations.[169]

Noise at night is known to cause a variety of sleep disturbances, including delay of sleep onset, overt awakening, brief arousals seen on EEG, changes in length and timing of sleep stages, and premature final awakening. Short-term effects of noise during sleep include noise-induced body movements and modifications of autonomic functions such as heart rate, blood pressure, vasoconstriction, and respiratory rate. Noise-induced body movements indicate a low level of arousal from sleep, and occur with noise events as low as 32 dBA. Arousals detected by brain wave pattern on EEG occur with noise events as low as 35 dBA, and conscious awakenings with events of 42 dBA.[170]

Much of the extensive literature on community noise and sleep disturbance focuses on neuroendocrine changes in catecholamine and cortisol levels due to noise disturbance,[171] short-term changes in circulation, including blood pressure, heart rate, cardiac output, and vasoconstriction,[172,173] and the effects of long-

---

169 Grimm et al. 1989.

170 Muzet A, Miedema H. 2005. Short-term effects of transportation noise on sleep with specific attention to mechanisms and possible health impact. Draft paper presented at the Third Meeting on Night Noise Guidelines, WHO European Center for Environment and Health, Lisbon, Portugal, April 26–28. Pp. 5–7 in *Report on the Third Meeting on Night Noise Guidelines*, available at www.euro.who.int/Document/NOH/3rd_NNG_final_rep_rev.pdf.

171 Ising H, Braun C. 2000. Acute and chronic endocrine effects of noise: review of the research conducted at the Institute for Water, Soil and Air Hygiene. Noise Health 7: 7–24.

172 Babisch W. 2003. Stress hormones in the research on cardiovascular effects of noise. Noise Health 5(18): 1–11.

173 Babisch W. 2005. Guest editorial: Noise and health. Environ Health Perspect 113(1): A14–15.

term exposure on the risk of myocardial infarction.[174] There is a significant exposure-response relationship between exposure to nighttime aircraft noise, daily average road traffic noise, and hypertension.[175–177]

Most studies of sleep do not differentiate between low frequency and other types of noise, but there is a growing awareness of the particularly disturbing nature of the low frequency components of community noise.[178] One study compared children sleeping with heavy trucks passing two meters from the house walls every two minutes all night long, to children sleeping with traffic noise without the low frequency component. The low frequency noise–exposed children showed increased cortisol production during the first half of the night (an alteration in the normal circadian rhythm of secretion) compared to the other children.[179] Increased cortisol during the first half of the night was significantly related to restless sleep and difficulties in returning to sleep after awakening during the night.

---

[174] Babisch W, Beule B, Schust M, Kersten N, Ising H. 2005. Traffic noise and risk of myocardial infarction. Epidemiology 16(1): 33–40.

[175] Jarup L, Babisch W, Houthuijs D, Pershagen G, Katsouyanni K, Cadum E, Dudley M-L, Savigny P, Seiffert I, Swart W, Breugelmans O, Bluhm G, Selander J, Haralabidis A, Dimakopoulou K, Sourtzi P, Velonakis M, Vigna-Taglianti F. 2008. Hypertension and exposure to noise near airports: the HYENA study. Environ Health Perspect 116(3): 329–33.

[176] Eriksson C, Rosenlund M, Pershagen G, Hilding A, Ostenson C-G, Bluhm G. 2007. Aircraft noise and incidence of hypertension. Epidemiology 18(6): 716–21.

[177] Haralabidis AS, Dimakopoulou K, Vigna-Taglianti F, Giampaolo M, Borgini A, Dudley M-L, Pershagen G, Bluhm G, Houthuijs D, Babisch W, Velonakis M, Katsouyanni K, Jarup L. 2008. Acute effects of night-time noise exposure on blood pressure in populations living near airports. European Heart J 29(5): 658–64.

[178] Persson Waye K. 2004. Effects of low frequency noise on sleep. Noise Health 6(23): 87–91.

[179] Ising H, Ising M. 2002. Chronic cortisol increases in the first half of the night caused by road traffic noise. Noise Health 4: 13–21.

706

Low frequency noise

Birgitta Berglund, lead editor of the WHO *Guidelines for Community Noise*,[180] stated in a review of low frequency noise effects:

> Although the effects of lower intensities of low-frequency noise are difficult to establish for methodological reasons, evidence suggests that a number of adverse effects of noise in general arise from exposure to low frequency noise: Loudness judgments and annoyance reactions are sometimes reported to be greater for low-frequency noise than other noises for equal sound-pressure level; annoyance is exacerbated by rattle or vibration induced by low-frequency noise; speech intelligibility may be reduced more by low-frequency noise than other noises except those in the frequency range of speech itself, because of the upward spread of masking.
>
> Low-frequency noise (infrasound included) is the superpower of the frequency range: It is attenuated less by walls and other structures; it can rattle walls and objects; it masks higher frequencies more than it is masked by them; it crosses great distances with little energy loss due to atmospheric and ground attenuation; ear protection devices are much less effective against it; it is able to produce resonance in the human body; and it causes greater subjective reactions (in the laboratory and in the community studies) and to some extent physiological reactions in humans than mid- and high frequencies.[181]

[180] World Health Organization. 1999. *Guidelines for Community Noise*, ed. Berglund B, Lindvall T, Schwela DH. 159 pp. www.who.int/docstore/peh/noise/guidelines2.html

[181] Berglund B, Hassmen P, Job RFS. 1996. Sources and effects of low frequency noise. J Acoust Soc Am 99(5): 2985–3002, p. 2985.

> Low-frequency noise also differs from other noise in producing vibrations of the human body and other objects.... Motion sickness has been linked to low-frequency noise even without accompanying vibration.[182]

Many subjects in the present study stated that turbine noise was different from other types of noise, using words like "invasive" and "unnatural," and saying that it was impossible to get used to this noise. Several said it wouldn't sound loud to people who did not live at their homes, or they described a "swish" or "hum" as extremely bothersome noises. A number spoke favorably of living near heavily traveled roads or urban train lines, compared to living near wind turbines. All who moved, moved into villages, towns, or suburbs, where there was more traffic but no danger of turbines being built next to them. The descriptions make it clear that there is a disturbing quality about turbine noise which is more than its audible loudness and that, over time, people become sensitized to wind turbine noise, rather than get used to it.

In the present study, Mr. and Mrs. G described a resonance or standing wave phenomenon in one room of their turbine-exposed home. At one end of this room, Mrs. G felt internal vibration, even though she could not feel any surfaces or objects vibrating when she put her hand on them. Mr. G felt peculiar in the same place, and always had to walk quickly away from that spot before his feeling progressed to nausea. In the home of family C, an audiologist detected vibration in the floor of a small room the family identified as having the worst problem in the home, and felt nauseated when he put his forehead against it.[183]

---

182 Berglund et al. 1996, p. 2993.

183 Personal communication from acoustician; name withheld for confidentiality reasons.

708

At a NASA test facility in the 1960's, healthy young men were exposed to low frequency noise in the 1–50 Hz frequency range at 110 to 150 dB for 2–3 minutes (high amplitude and short duration). Over the full 1–50 Hz frequency range they experienced fatigue and took longer to perform assigned tasks. At frequencies less than 25 Hz there was an "annoying tickling" in the ear. In the same frequency range, there were modulations of speech, moderate vibrations of the chest, and fullness in the hypopharynx with an annoying gag sensation. "In regard to the opinions of those tested, it was indicated that the sensations involved were impressive."[184]

A case that was similar to the cases presented in this paper involved a couple in Germany in 1996. After moving into a new house outside a provincial city, the couple experienced symptoms with increasing intensity, including "indisposition, decrease in performance, sleep disturbance, headache, ear pressure, crawl parasthesy,[185] or shortness of breath."[186] Their case was intensely investigated with both A-weighted and linear measurements of noise indoors and outdoors, correlated in real time with the couple's symptoms. In time, the symptoms were correlated with intensity of noise below 10 Hz. The couple's symptoms and the intensity of noise below 10 Hz both varied with the wind and weather, and were worse in the winter. No plausible mechanism for production of such noises or correspondences to local sources of noise, such as the housing complex heating plant, was found. Symptoms occurred when the sound pressure level at 1 Hz was 65 dB, well

---

[184] Edge PM, Mayes WH. 1966. Description of Langley low-frequency noise facility and study of human response to noise frequencies below 50 cps. NASA Technical Note, NASA TN D-3204. 11 pp.

[185] *Paresthesia* means a prickling sensation, the "pins and needles," felt when a numb foot is waking up. I interpret "crawl parasthesy" to mean a sensation like insects crawling on the skin or in the chest. One of the current study's subjects, I2, also described "pins and needles" inside her chest.

[186] Feldmann J, Pitten FA. 2004. Effects of low-frequency noise on man: a case study. Noise Health 7(25): 23–28.

below hearing threshold. None of the frequencies responsible for the symptoms, all below 10 Hz, had sound pressure levels above 80 dB. The decibel levels that affected the man and wife in their home were far less than their own threshold hearing levels measured in a sound lab. The authors hypothesized that infrasound, with its very long wavelengths (10 Hz, for example, has a 34 m wavelength in air), causes strong pressure fluctuations in relatively small closed rooms—pressure fluctuations that are detected more by the whole body and its inner organs than by the ears.

Similar intensive investigations, using linear as well as A-weighted sound levels, 1/3 octave sound pressure levels down to 1 Hz, indoor measurements, and assessments of wall vibration, have proved fruitful in other low frequency noise complaint investigations.[187] These investigators, from a state environmental agency in Germany, paid attention to spontaneous statements by the affected people, to see whether perceptions of noise followed a systematic pattern. They found that "noises which in many cases induced vehement complaints were to a large extent of rather low sound levels,"[188] and that indoor ventilator noise and noises generated by structure-borne sound transmission were distinctly more disturbing than road traffic noise. These authors documented standing waves in rooms by measuring and comparing loudness in dBA and dB(lin) at the center of the room and near walls. They detected vibration in walls, and correlated the dominant frequency and its corresponding wavelength to the size of the room in discussing how a standing wave was established in the room.

For this kind of complaint, the authors noted,

[187] Findeis H, Peters E. 2004. Disturbing effects of low-frequency sound immissions and vibrations in residential buildings. Noise Health 6(23): 29–35.

[188] Findeis and Peters 2004, p. 29.

710

> More than half . . . were made on the grounds of sleep disturbance. Quite often symptoms like "a roaring in the head, especially when lying down" were brought forward. Time and again, "a feeling of riding a lift [elevator]" was reported, and over and again the measuring team had the impression that the reported immissions [noise] meant a nerve-wracking experience for the exposed persons. Several complainants even got into a state of being aggressive. There were reports by a number of trustworthy persons on how they at first—for instance when moving into the flat—did not even notice any immissions. But in the course of a few weeks they began to perceive them distinctly and [the immissions] became intolerable after continued exposure. It was obvious that in these cases the sensibility of specific noise components had developed. Thus, it is understandable that non-exposed persons were at a difficulty to even acknowledge such noise immissions.[189]

Wind turbines produce noise in the low and infrasonic frequency ranges. The issue has not been whether they produce low frequency or infrasonic noise, but whether the amplitudes are sufficient to cause human effects. According to data published by van den Berg,[190] unweighted amplitudes at 1 Hz, at one wind park under one set of weather conditions, were in the 70–100 dB range, declining to the 55–75 dB range at 10 Hz and the 50–60 dB range at 100 Hz. Wind turbine noise has a pulsating quality, produced as the airfoil blades swing past the tower, compressing the air between blade and tower. These low frequency pressure fluctuations, among other effects, modify the loudness of the higher frequency sounds coming from the turbines, producing the audible "swish"

[189] Findeis and Peters 2004, p. 32.

[190] van den Berg 2004a.

that synchronizes with the feeling of pulsation some subjects felt in their chests. Coming from several towers at once, these low frequency air pressure fluctuations may synchronize and reinforce, depending on the orientation of the towers and house and the timing of the individual turbines. Three families in this study (A, B, and F) lived in houses nearly in line with a row of turbines. For families A and B, the area's worst storms, "nor'easters," swept right down the line towards their houses, which were built on a hill at the level of the turbine hubs. These two families, though they were a kilometer (about 3300 feet) from the closest of the 10 turbines, moved out faster—in five months—than any of the other families, and had particularly severe symptoms.

Studies of turbine noise also show that noise carries farther than predicted by conventional industry modeling. This has to do not only with the low frequency components of the noise, which attenuate less with distance, but also with layering of the atmosphere at night, which creates cool still air at ground level and brisk, laminar airflow at turbine hub heights.[191] Industry models do not take these factors into account. Nor do they allow for a noise source more than 30 m above the ground. (Turbine hub heights in this study were 59–90 m.) Nor do they allow for increased transmission of sound in front of and behind the blades (with less sound transmission in the plane of the blades, including under the turbines), sky reflections, or weather conditions that focus the noise transmissions.[192]

Vibroacoustic Disease (VAD) model

High intensities of low frequency noise over prolonged time periods may cause marked neurologic damage, as described

---

[191] van den Berg 2004b.

[192] Richard James, INCE Full Member, personal communication, 5/11/08.

that synchronizes with the feeling of pulsation some subjects felt in their chests. Coming from several towers at once, these low frequency air pressure fluctuations may synchronize and reinforce, depending on the orientation of the towers and house and the timing of the individual turbines. Three families in this study (A, B, and F) lived in houses nearly in line with a row of turbines. For families A and B, the area's worst storms, "nor'easters," swept right down the line towards their houses, which were built on a hill at the level of the turbine hubs. These two families, though they were a kilometer (about 3300 feet) from the closest of the 10 turbines, moved out faster—in five months—than any of the other families, and had particularly severe symptoms.

Studies of turbine noise also show that noise carries farther than predicted by conventional industry modeling. This has to do not only with the low frequency components of the noise, which attenuate less with distance, but also with layering of the atmosphere at night, which creates cool still air at ground level and brisk, laminar airflow at turbine hub heights.[191] Industry models do not take these factors into account. Nor do they allow for a noise source more than 30 m above the ground. (Turbine hub heights in this study were 59–90 m.) Nor do they allow for increased transmission of sound in front of and behind the blades (with less sound transmission in the plane of the blades, including under the turbines), sky reflections, or weather conditions that focus the noise transmissions.[192]

Vibroacoustic Disease (VAD) model

High intensities of low frequency noise over prolonged time periods may cause marked neurologic damage, as described

---

[191] van den Berg 2004b.

[192] Richard James, INCE Full Member, personal communication, 5/11/08.

 713

by the Vibroacoustic Disease (VAD) group in Portugal.[193] This is a provocative body of research, full of interesting case descriptions and pathology studies, but compromised by absence of specified study group criteria, absence of control groups, and lack of quantification. The study group consists of 140 aircraft maintenance and repair technicians in the Portuguese Air Force, of whom 22 (15.7%) had adult-onset epilepsy, compared to a national prevalence of 0.2%.[194] Some of the case descriptions of the subjects with epilepsy also include cognitive decline, depression, paranoia, and rage attacks.[195] The descriptions are similar to those of retired professional football players with histories of multiple concussions.[196,197] The vibroacoustic disease researchers ascribe VAD pathology to whole-body vibration induced by the noise, with the pathology of each body part induced by vibration of that part. Neurologic effects may be due to neuronal or axonal shearing, as in the multiple concussions scenario, or due to microangiopathy in the brain, meaning, effects on and occlusion of small blood vessels.[198]

With regard to the chest, the VAD researchers have used human autopsy and biopsy and animal rearing studies to describe loss of

---

[193] Castelo Branco and Alves-Pereira 2004.

[194] Castelo Branco and Alves-Pereira 2004.

[195] Martinho Pimenta AJ, Castelo Branco NAA. 1999. Neurological aspects of vibroacoustic disease. Aviat Space Environ Med 70(3): A91–95.

[196] Omalu BI, DeKosky ST, Minster RL, Kamboh MI, Hamilton RL, Wecht CH. 2005. Chronic traumatic encephalopathy in a National Football League player. Neurosurgery 57: 128–34.

[197] Omalu BI, DeKosky ST, Hamilton RL, Minster RL, Kamboh MI, Shakir AM, Wecht CH. 2006. Chronic traumatic encephalopathy in a National Football League player: part II. Neurosurgery 59: 1086–93.

[198] Martinho Pimenta and Castelo Branco 1999.

714

cilia and microvilli from epithelial surfaces of the bronchi,[199–201] pleura,[202] and pericardium.[203] They also describe thickening of bronchial epithelial basement membrane,[204] pericardium,[205] and blood vessel walls[206] by extra, organized collagen and elastin. Several of the animal-rearing studies on bronchial epithelial changes are well controlled and convincing.[207,208]

Based on the vibroacoustic disease research, I hypothesize that vibratory or pulsating air pressure fluctuations in subjects' airways in the present study may induce shearing of surface cilia, thus impairing the clearance of mucus and particulates from airways. This in turn could make subjects more susceptible to lower respiratory infections and increased airway irritation and reactivity (asthma). The Eustachian tube and middle ear could be susceptible

---

199 Oliveira MJR, Pereira AS, Ferreira PG, Guinaraes L, Freitas D, Carvalho APO, Grande NR, Aguas AP. 2004. Arrest in ciliated cell expansion on the bronchial lining of adult rats caused by chronic exposure to industrial noise. Environ Res 97: 282–86.

200 Oliveira MJR, Pereira AS, Castelo Branco NAA, Grande NR, Aguas AP. 2002. In utero and postnatal exposure of Wistar rats to low frequency/high intensity noise depletes the tracheal epithelium of ciliated cells. Lung 179: 225–32.

201 Monteiro M, Ferreira JR, Alves-Pereira M, Castelo Branco NAA. 2007. Bronchoscopy in vibroacoustic disease I: "pink lesions." Inter-Noise 2007, August 28–31, Istanbul, Turkey.

202 Pereira AS, Grande NR, Monteiro E, Castelo Branco MSN, Castelo Branco NAA. 1999. Morphofunctional study of rat pleural mesothelial cells exposed to low frequency noise. Aviat Space Environ Med 70(3): A78–85.

203 Castelo Branco NAA, Aguas AP, Pereira AS; Monteiro E, Fragata JIG, Tavares F, Grande NR. 1999. The human pericardium in vibroacoustic disease. Aviat Space Environ Med 70(3): A54–62.

204 Castelo Branco NAA, Monteiro M, Ferreira JR, Monteiro E, Alves-Pereira M. 2007. Bronchoscopy in vibroacoustic disease III: electron microscopy. Inter-Noise 2007, August 28–31, Istanbul, Turkey.

205 Castelo Branco et al. 1999.

206 Castelo Branco NAA. 1999. A unique case of vibroacoustic disease: a tribute to an extraordinary patient. Aviat Space Environ Med 70(3): A27–31.

207 Oliveira et al. 2004.

208 Oliveira et al. 2002.

to the same process, leading to prolonged middle ear effusions and unusual acute infections.

The increased asthma seen in subjects F1 and F3 may also have a connection to their frequent use of paracetemol (acetaminophen) for headaches during turbine exposure.[209]

Community noise studies and *annoyance*

Studies of community noise frequently assess a quality called *annoyance*. "Apart from 'annoyance,'" the World Health Organization writes, "people may feel a variety of negative emotions when exposed to community noise, and may report anger, disappointment, dissatisfaction, withdrawal, helplessness, depression, anxiety, distraction, agitation, or exhaustion."[210]

Beyond even these negative emotions, moving out of an owned home indicates that people feel sick and under threat, judging that their survival and well-being, and that of their children, will be enhanced by moving out—even as they exhaust limited resources to do so and face unrecompensed loss of their major asset, their home.

Sick and *annoyed* are not the same thing. In English, *annoyance* carries an air of triviality, like a mosquito buzzing around one's head. *Sickness* threatens survival itself.

Pedersen and Persson Waye assessed annoyance (which may be a shorthand for the above list of negative emotions, but remains different from sickness) among 351 households near wind turbines in Sweden in 2000. They used a mailed survey and compared annoyance to modeled A-weighted sound pressure levels they

---

[209] Beasley et al. 2008.

[210] World Health Organization 1999, *Guidelines for Community Noise*, p. 50.

716

calculated to exist outside homes near clusters of one to five turbines of power 0.15–0.65 MW (much smaller than in the current study), based on the homes' distances from turbines.[211] They found people to be highly annoyed by wind turbine noise at sound pressure levels much lower than for other types of community noise. The A-weighted decibel level (in a measure averaged and weighted over time, L_{eq}) that corresponded to 15% of the people being highly annoyed was 38 dBA for wind turbines, 57 dBA for aircraft, 63 dBA for road traffic, and 70 dBA for railways. The curve for annoyance due to wind turbine noise had a steep slope, so that by 41 dBA, 35% of people were *highly annoyed*. Sixteen percent of respondents over 35 dBA reported that their sleep was disturbed by wind turbine noise.

I interpret this result as an indication of the degree to which wind turbine noise has a disturbing quality not captured by its A-weighted measurement. Since A-weighting emphasizes higher frequencies and filters out lower frequencies, the qualitative difference may be related to the presence of low frequency components. Even without directly measuring the low frequency components, this study is potentially useful with regard to regulating noise and determining setback distances for turbines. Since the study was done in units of dBA outside houses, and most community noise regulations (including for wind turbines) also use units of dBA outside houses, we can easily translate this result into the recommendation that wind turbine ordinances need to limit the turbine noise levels outside houses to less than 35 dBA. This does not mean that only 35 dB of real noise is present, but rather that in the common measurement unit of community noise—which is dBA—35 is a number that represents a significant amount

[211] Pedersen E, Persson Waye K. 2004. Perception and annoyance due to wind turbine noise: a dose-response relationship. J Acoust Soc Am 116(6): 3460–70.

717

of sleep disturbance and high annoyance if the noise comes from wind turbines.

In a continuation study that involved interviewing participants, Pedersen found that some people had moved out of their homes, rebuilt their homes in an attempt to exclude turbine noise, or begun legal proceedings because of problems associated with turbine exposure.[212] Pedersen and Persson Waye also found informants who were sensitive to both noise and blade motion, felt violated or invaded by turbine noise, and found their houses to be places where they could no longer find restoration[213]—qualitative similarities to the current study.

Van den Berg, Pedersen, and colleagues conducted another survey study of noise and annoyance in the Netherlands in 2007.[214] They mailed questionnaires to 1960 households within 2.1 km (1.3 mi) of at least two adjacent 0.5–3 MW turbines, with 725 responses (37% response rate). The questionnaire asked about visual and auditory perceptions, economic benefit, annoyance, chronic diseases, current symptoms, psychological stress, and sleep disturbance, and looked at variation in these factors (as in the Swedish study) against modeled A-weighted noise levels.

Though it contained several questions about health, this study was not properly constructed to sample health in an accurate or realistic way. The evidence for this is found in the study results themselves, which contain significant bias or skew relative to known health parameters.

---

212 Pedersen 2007.

213 Pedersen and Persson Waye 2007.

214 van den Berg et al. 2008b.

718

For example, 2% of respondents in this study indicated that they had chronic migraine disorder.[215] The population prevalence of migraine disorder is remarkably stable across countries and time when controlled for age, sex, and definition of the disease, being 5–6% for males and 15–18% for females.[216,217] A finding of 2% is an underestimate, indicating that something about this study's method of sampling migraine prevalence was awry.

Sampling and sampling error occur at several levels, such as the level of selecting respondents and the level of sampling the respondents' thoughts through questioning. Potential flaws at each level can be identified in this study.

First, the researchers attempted to elicit objective health information with just two questions in this survey, one on past or underlying health and one on current symptoms. (Separate questions addressed sleep disturbance.) This is the single question about underlying health:

> 37. Do you have any long term/chronic disease? (no → 38, yes). *If yes, which chronic disease do you have?* (diabetes, high blood pressure, tinnitus, hearing impairment, cardiovascular disease, migraine, other *viz:*)[218]

This is a very brief and superficial question, and it is not surprising that it failed to capture all the diagnoses of migraine that should have been present in a random population sample. In medical

---

[215] van den Berg et al. 2008b, p. 48.

[216] Lipton RB, Bigal ME, Diamond M, Freitag F, Reed ML, Stewart WF; AMPP Advisory Group. 2007. Migraine prevalence, disease burden, and the need for preventive therapy. Neurology 68(5): 343–49.

[217] Stewart WF, Simon D, Shechter A, Lipton RB. 1995. Population variation in migraine prevalence: a meta-analysis. J Clin Epidemiol 48(2): 269–80.

[218] van den Berg et al. 2008b, Appendix p. 5.

719

research, in contrast, the presence or absence of a diagnosis in a subject is established by multiple proven and validated questions directly tied to the formal definition of the illness, administered by a trained interviewer. Even in clinical practice, which is less formal, an accurate review of systems still requires a series of specific screening questions and the knowledge of when and how to question in further depth. No clinician or health researcher would rely on a question like the above to elicit full and accurate information about the past health history.

The same question also failed to elicit accurate prevalence figures for tinnitus. Tinnitus prevalence among survey respondents was 2%, whereas 4% is the likely population-level figure for the respondents' average age of 54.[219] Tinnitus prevalence also did not show age differences in this sample,[220] whereas in reality tinnitus has a well-documented pattern of increasing prevalence with advancing age.[221]

The question's time frame is also unclear. Were the authors trying to find out about baseline susceptibilities (health conditions before turbines) or did they hypothesize that exposure to wind turbines might alter the prevalence of these chronic conditions? Though they never state it explicitly, their analysis makes it clear they hypothesized that health effects due to wind turbines, if they exist, would present as higher levels of the listed chronic diseases closer to wind turbines.[222] To think that they might find such an effect with this type of sample size and mode of study verges on silly, it is

[219] National Institute on Deafness and Other Communication Disorders, USA, website, "Prevalence of chronic tinnitus." 2009. www.nidcd.nih.gov/health/statistics/prevalence.htm

[220] van den Berg et al. 2008b, p. 47.

[221] National Institute on Deafness and Other Communication Disorders, "Prevalence of chronic tinnitus." 2009.

[222] van den Berg et al. 2008b, p. 50.

so far outside the parameters of how such issues are studied (see, for example, studies cited in footnotes 171–177, above). As a result, this study's failure to find such an effect is meaningless.

There were also sampling problems at the level of subject selection. First, the study has no control population that is not exposed to turbine noise. It samples within 2.1 km (1.3 mi) of turbines, using the unspoken assumption that the people at the outer edge of this radius will not be exposed to significant amounts of turbine noise and can therefore act as a control group. An epidemiologic study, in contrast, would have a control group of households subjected to all the same procedures for household selection, questioning, and noise modeling as the study group, but without turbines present.

Second, uncontrolled subject selection processes occurred at the level of the household. Once questionnaires reached households, what happened? Nearly two-thirds of households declined to respond. The researchers studied a subset of non-responders using a very brief questionnaire that yielded a modestly higher (48%) response rate. The brief questionnaire showed that non-responders were similar to responders in their average degree of annoyance at wind turbine noise, but did not address the issue of whether non-responders differed from responders in health parameters.

An additional process of self-selection occurred within responder households, since only one individual replied and only answered questions about himself. The householders chose who replied. On a very mundane and human level, we can imagine how this process might have selected against migraineurs in the sample, if the person with a headache the day the survey arrived asked someone else to fill it out.

The survey's second question about health concerned current symptoms, as follows:

721

38. Have you been troubled by the following symptoms during the last months? ((almost) never, at least once a month, at least once a week, (almost) daily) [sic]

Headache
Undue tiredness
Pain and stiffness in the back, neck or shoulders
Feeling tense or stressed
Depressivity
Not very sociable, wanting to be alone
Irritable
Resigned
Fearful
Concentration problems
Nausea
Vertigo
Mood changes
Other, namely: *(please indicate what)*[223]

This is an odd list of "symptoms"—an undifferentiated mix of physical and psychological, with a few simple "feeling words" thrown in. It does not make sense as a symptom list—not without more detail and structuring into symptom groups. As with the chronic disease question, above, medical researchers and clinicians know that accurate and complete information cannot be elicited in this format, especially about delicate subjects like mood states and health. This question, too, is unclear about timing—pre-existing vs. during exposure, while near turbines or away from them.

This question in fact yielded little information that was useful to the researchers. In their analysis, the only reference to the health symptoms question is as follows:

[223] van den Berg et al. 2008b, Appendix p. 6.

723

> Respondents who did not benefit economically from wind turbines reported more chronic diseases and health symptoms than those who benefited.... The observed differences between the sub-samples regarding chronic diseases and health symptoms could be due to age effects; respondents who did not benefit economically were older than those who benefited.[224]

Otherwise, through a long and detailed statistical analysis of stress, sleep disturbance, noise, annoyance, and chronic disease, the health symptoms question does not appear again.

The researchers expanded their questioning on mood states by incorporating a screening interview for mental illness used in general medical practice, called the General Health Questionnaire.[225] Despite the name, it is not a health questionnaire, nor is it a measure of psychological stress (which is how the authors use it). The GHQ-12 is a screening tool for mental illness, used to help a physician figure out which of his presenting patients need assessment for psychiatric illness. It was validated (meaning compared against other effective means of diagnosis to see if it identified the right people) for its declared purpose, not as a measure of psychological stress. The authors present it as a "validated instrument" for "measuring 'perceived health,'"[226] then use it in their analysis as a measure of "psychological stress," morphing the question set from one purpose to another to another without justification.[227]

---

[224] van den Berg et al. 2008b, p. 49.

[225] Goldberg DP, Hillier VF. 1979. A scaled version of the General Health Questionnaire. Psychol Med 9(1): 139–45. The 28-item GHQ may be found at http://www.gp-training.net/protocol/docs/ghq.doc and the 12-item GHQ (used by van den Berg et al.) at www.webpoll.org/psych/GHQ12.htm.

[226] van den Berg et al. 2008b, p. 20.

[227] van den Berg et al. 2008b, p. 47.

In the Dutch survey study results, owners of turbines lived the closest to turbines and were able to turn them off if they or their neighbors were bothered by the noise—a key difference between the Netherlands and other countries. These closer respondents tended to be farmers and to benefit economically from the turbines. They were on average younger, healthier, and, as it happens, better educated than the respondents living farther from turbines.

Sleep disturbance, annoyance, and questionnaire measures of stress were correlated with noise levels among people who did not benefit economically from turbines. Annoyance occurred at lower dBA noise levels than for road, rail, or air traffic noise, as in the similar Swedish study. Being awakened from sleep was associated with higher noise levels, and difficulty falling asleep and higher stress scores were associated with annoyance. "Respondents with economic benefits reported almost no annoyance,"[228] though they lived closest to the turbines and experienced the highest modeled noise levels. If turbine owners were turning the turbines off when they were bothered or during sleep, then the modeled noise levels would not have accurately represented real noise levels close to the turbines.

Despite health being inadequately sampled in this study, the authors still draw conclusions that are interpreted popularly as evidence against health effects by wind turbines, in sentences like this one from the authors' summary: "There is no indication that the sound from wind turbines had an effect on respondents' health, except for the interruption of sleep."[229] Though it is downplayed in this sentence, sleep interruption is in fact of great significance to health. The authors are remiss in failing to acknowledge that the study methods do not have the power to detect other health effects.

---

[228] van den Berg et al. 2008b, Summary, p. ii.

[229] van den Berg et al. 2008b, Summary, p. ii.

724

The authors would have more accurately captured the survey's health results had they written, "Sleep disturbance or interruption, an effect of profound importance to health, was correlated with turbine noise levels. Unfortunately, the survey could not effectively address other health questions due to bias introduced at the level of data collection. An important finding is the possibility of biased responses from respondents benefiting economically from turbines, yet it is equally possible that turbine owners are in the habit of turning turbines off at critical times, thus avoiding both annoyance and sleep disturbance."

Recommendations

For physicians practicing near wind turbine installations, I suggest incorporating proximity to turbines into the personal and social history in a neutral and non-suggestive way, especially for the types of symptoms described in this report.

With regard to turbine setback from dwellings: in Table 1B we see that the subjects in the current study lived between 305 m (1000 ft) and 1.5 km (4900 ft or 0.93 mi) from the closest turbine. There were three severely affected families at 930–1000 m (3000–3300 ft) from turbines. This study suggests that communities that allow 305–457 m (1000–1500 ft) setbacks from homes, like those in New York State, may have families who need to move after turbines go into operation.

All turbine ordinances, I believe, should establish mechanisms to ensure that turbine developers will buy out any affected family at the full pre-turbine value of their home, so that people are not trapped between unlivable lives and destitution through home abandonment. By shifting the burden of this expense to turbine developers, I would hope that developers might have a stronger incentive to improve their techniques for noise prediction and to accept noise level criteria recommended by such agencies as the World Health Organization and the International Standards Organization,[230] and fortified by the findings of Pedersen (above).

With regard to families already affected, developers and permitting agencies share the responsibility for turbines built too close to homes, and together need to provide the financial means for these families to re-establish their lives at their previous level of health, comfort, and prosperity.

I support the recommendations for noise level criteria and procedures for noise monitoring by George Kamperman and Richard James.[231] A single setback distance may not be both protective and fair in all environments with all types of turbines, but it is clear, from the current study and others, that minimum protective distances need to be more than the 1–1.5 km (3280–4900 ft or 0.62–0.93 mi) at which there were severely affected subjects in this study, more than the 1.6 km (5250 ft or 1 mi) at which there were affected subjects in Dr. Harry's UK study,[232] and, in mountainous terrain, more than the 2–3.5 km (1.24–2.2 mi) at which there were symptomatic subjects in Professor Robyn Phipps's New Zealand study.[233]

Two kilometers, or 1.24 miles, remains the baseline shortest setback from residences (and hospitals, schools, nursing homes, etc.) that communities should consider. In mountainous terrain, 2 miles (3.2 km) is probably a better guideline.

---

[230] See Kamperman and James 2008b.

[231] Kamperman and James 2008b. Presented in shorter form, Kamperman GW, James RR. 2008a. Simple guidelines for siting wind turbines to prevent health risks. Noise-Con, July 28–31, annual conference of the Institute of Noise Control Engineering/USA.

[232] Harry 2007.

[233] Phipps 2007.

726

Setbacks may well need to be longer than these minima, as guided by the noise criteria developed by Kamperman and James.

Suggestions for further research

- Epidemiologic studies comparing populations exposed and not exposed to wind turbines with regard to the prevalence of specific symptoms, such as tinnitus and balance complaints. Such studies might be best conducted in European countries that have both national health data systems and significant numbers of wind turbines.
- Case series by neurotologists internationally, who are able to do appropriate objective examinations and testing in addition to clinical history.
- Collaboration between physicians and independent noise engineers to find which specific frequencies and intensities of sound and vibration correlate with subjects' symptoms in real time, and to establish a standard protocol for wind turbine noise sampling that includes these specific frequencies and intensities of sound and vibration.
- Further clinical/laboratory research on the effects of low frequency noise and vibration on the human vestibular system.
- Case control studies by specialist physicians near turbine installations on rarer associated symptoms, such as ocular problems, lower respiratory infections, asthma, persistent middle ear effusions, failure of anticoagulation, loss of diabetes control, exacerbation of arrhythmias, and exacerbation of gastrointestinal conditions.
- Studies of turbine noise and children's learning. Standardized test scores, before and after turbines are built near schools or in a community, might be compared to test scores of similar,

non-exposed schools and communities across the same years. The current study suggests that both school and home turbine noise exposures would have to be quantified.

Limitations of the study

- The study was done by interview and only limited medical records were available. Physical exam and appropriate testing (such as hearing, balance, and neuropsychological testing) would clarify and provide objective evidence for otologic and neurologic problems. Physical exam and appropriate testing are necessary to assess the rarer associated conditions not included in the core symptoms of Wind Turbine Syndrome.
- Participant memory limitations or distortions. I excluded several families from the analysis because they were unclear about what had happened when, combined with not having spent enough time in a post-exposure situation. I insisted on a post-exposure period to compensate for the difficulty of accurately comparing before-exposure experience to the current situation of exposure.
- Minimization or exaggeration of effects. I felt some subjects may have minimized potentially embarrassing or frightening issues, such as nocturia in men and cognitive difficulties in general. In other families, excluded from the analysis, one spouse was clearly committed to staying in the house and minimized what the other spouse said. I endeavored to protect against exaggeration by including in the study only families who had moved out of their homes or done something else expensive in response to their symptoms, proving their symptom severity in ways other than words. The one exception to this rule was the family of an American physician and nurse, whose professionalism, I felt, was protective.

- The study was limited to English-speaking subjects. There was only one non-native speaker. He was competent at English and had an English-speaking wife, but there may have been subtleties in his symptoms that he didn't tell me about.
- Small case series sample. For this study, I chose a cluster of the most severely affected and most articulate subjects I could find. It is not a large enough sample to establish a gradient of effects with a gradient of exposure (distance from the turbines). It is not an epidemiologic sample that could establish prevalence of effects within exposure gradients or according to age or pre-existing conditions. Conditions that occurred in one or a few study subjects require case-control studies and cannot be established as part of the syndrome from this study.
- Limited duration of follow-up. For cognitive symptoms improved but not resolved at the post-exposure interview, the time course of resolution is not clear.